SOLVED:

THE RIDDLE OF HEART ATTACKS

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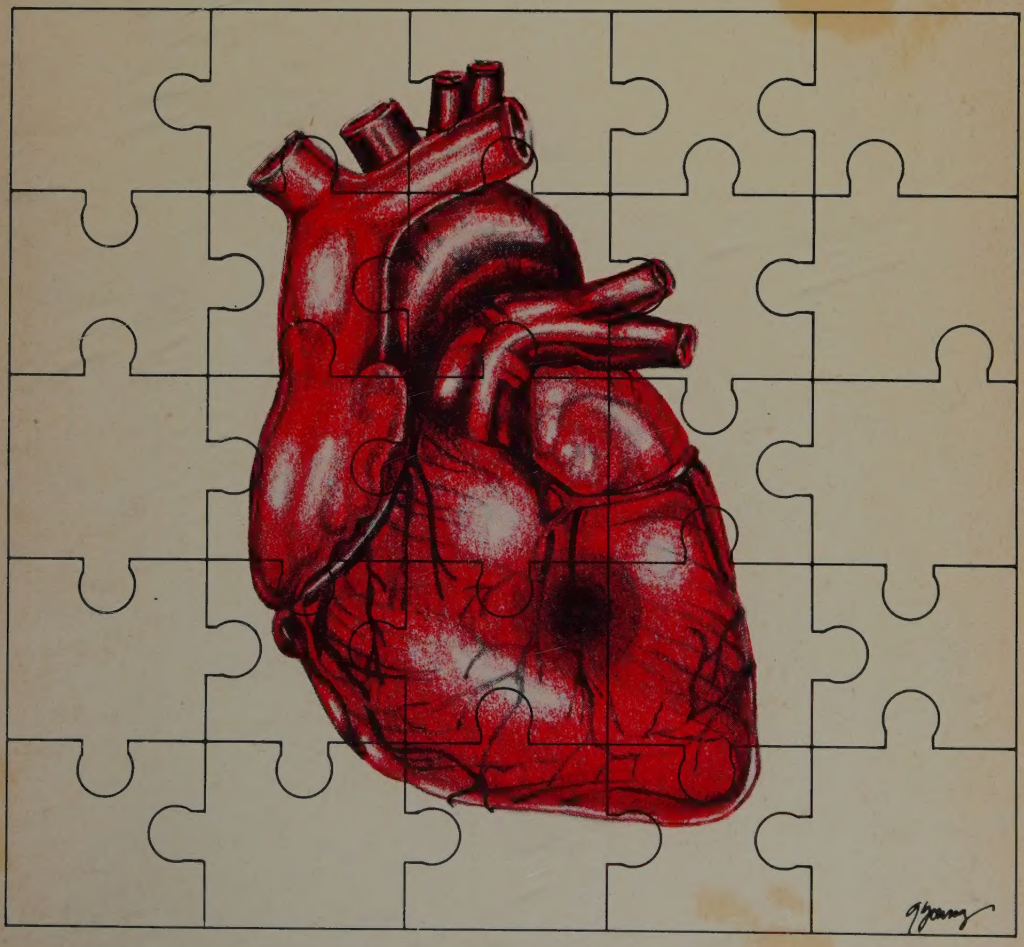

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Preface

The rapid rise in heart attacks during the 20th century has caused panic in the public and confusion among the scientists. Diet has received most of the blame, although cigarettes, a lack of exercise, and various environmental factors have also been suspected.

Autopsy studies clearly indicate that the major factor is a change in man himself. Eliminating deaths from infectious diseases at an early age has led to a NEW POPULATION of adults never seen before. Those escaping premature deaths from infections are highly susceptible to hardening of the arteries and to heart attacks.

Animals deprived of their thyroid glands show both a susceptibility to infections and hardening of the arteries. Administration of thyroid to such animals prevents damage to the arteries.

In man, those individuals with symptoms of low thyroid function, including a subnormal body temperature, are prone to hardening of the arteries. Thyroid administration to such individuals has markedly reduced the incidence of heart attacks.

After presenting overwhelming evidence that thyroid deficiency is the culprit in atherosclerosis, the final chapter urges the abandonment of polyunsaturated fats in our diet. In both experimental animals and man, in addition to toxic symptoms, a rise in cancer has been reported after prolonged ingestion of polyunsaturated fats.

Biography

Broda O. Barnes was born April 14, 1906 in a log cabin on the side of a rocky slope in southern Missouri. His interest in research began as an undergraduate in the chemistry department at the University of Denver. He taught physiological chemistry for two years at Western Reserve University where he received a Master's degree in 1930. He earned a Doctorate degree in physiology in 1931 at the University of Chicago; for five years he taught physiology there. During this interval many publications on research on the glands of internal secretions appeared. He finished his Medical degree in 1937 at Rush Medical College, interned at Illinois Research Hospital, and began the practice of medicine. For two years he was Assistant Professor of Medicine at the University of Illinois, then became Chairman of the Health Education Department of the University of Denver. He was called to the military twice: 1943-46 and 1950-51. He became Professor Affiliate in the Department of Physiology at Colorado State University, 1963-68. In the practice of medicine his interest has been prevention rather than treatment alone. Although engaged in General Medicine for 40 years, the thyroid gland has always enjoyed the center of the stage. Over 100 publications in scientific journals and three books have summarized his endeavors.

Charlotte Webster Barnes received her A.B. and A.M. at Oberlin College in geology with graduate work at Bryn Mawr College and at the University of Chicago. From the time of their marriage in 1932 she deserted the fossils to help in the field of medicine.

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CHAPTER I

The Riddle is Solved

No longer is there a secret about the cause of the explosion of heart attacks in the 20th century nor about the manner in which they occur. Mother Nature may have a magic wand on the TV screen, but when she is disrobed in the laboratory, she is only another female performing her tasks in the most precise and efficient manner so characteristic of her sex. For the busy reader, lacking time for all the controversial theories, the bare facts are expressed in this chapter, and discussions are left to subsequent pages.

In the race among many diseases to terminate mankind's earthly existence, heart attacks were unknown at the turn of the century; today they are Public Enemy Number One. In the sports world, a new champion is crowned when he defeats all challengers in competition. Diseases compete in much the same way to eliminate mankind; periodically a new champion is recognized. A prizefighter, because of age, may lose his crown to a younger, more vigorous challenger. All of the diseases are under a threat of extinction from the progress of medical science. *The reduction in deaths from infectious diseases played a major role in the rise of heart attacks.*

For a century, tuberculosis had been "The Captain of Death" ruling the roost like an overgrown bully among a group of small boys. Tuberculosis had won the championship not as a result of its own superiority but because an obscure physician, Dr. Jenner, had found that smallpox could be prevented by vaccination. Smallpox had led the pack for many years, decimating babies and children at an early age. Its conquest had permitted a larger section of the population to live into an older age bracket. History leaves no doubt that tuberculosis was the statistic replacing smallpox.

During the last half of the 19th century, public health measures opened a counter-attack on tuberculosis. Bed-rest in a sanitarium not only conserved the energy of the victim allowing him to live longer, but

it also isolated the source of the infection, thereby preventing its spread to other people. The net result was that thousands of deaths deserted the old champion, tuberculosis, and for several years, he wobbled on weak knees. However, he was not completely knocked out for the count of ten until the birth of the antibiotics. The major reason for the modern change in death patterns is the appearance of the antibiotics and not the result of changes in our environment.

It is well to bear in mind that tuberculosis became the champion because of the conquest of smallpox, a disease of babies and children. The eradication of smallpox allowed many babies to live into a more advanced age group, and tuberculosis happened to specialize in young adults. Hence, for a century the majority of deaths among young adults was due to tuberculosis; however, the average age of survival had gone up. Progress was being made.

The antibiotics stopped deaths from many infectious diseases, and the major conquest was in tuberculosis. Now the young adults by the millions began to live into another age bracket, the average age of survival making the biggest advance in history. This again indicated progress and not the end of the world. Heart attacks are a blessing in disguise; they can be eliminated any time merely by bringing back tuberculosis and lowering the average age of survival.

This may sound facetious, but it has actually happened. During World War II the incidence of heart attacks decreased precipitously in Europe. This was hailed as final proof by the diet faddists that cholesterol was the villain in heart attacks. Cholesterol foods were scarce during the war. Both observations are true; cholesterol foods were unavailable and heart attacks decreased, but the interpretation was 100 percent in error. The mistake was made because the "cholesterol activists" had not bothered to look at the protocols (records) of the autopsies carried out during the war, but had only counted those who had died from heart attacks.

I have personally reviewed 70,000 autopsy protocols at Graz, Austria, carried out between the years 1930-1970. Much more will be said later about this invaluable storehouse of knowledge; only the important facts will be presented here. At Graz, heart attacks dropped 75 percent between 1939 and 1945, and it is true that people were not eating cholesterol foods during the war. However, the low cholesterol diet did not protect their arteries from hardening. A look at the arteries of the entire series of 2000 autopsies in 1945 revealed that the number

of individuals with damage to their coronary arteries (arteries to the heart) was approximately doubled in 1945 compared to 1939, and the degree of damage to each one affected was about twice as great. In other words, the low cholesterol diet had not only failed to protect the arteries, but the damage was increased four-fold.

Then why had heart attacks dropped so suddenly? The answer was very simple, when all the causes of death were investigated. Tuberculosis had jumped more than heart attacks had decreased. This was the real reason for the drop in heart attacks; the patient was not living long enough. Furthermore, a careful look at the arteries in the hearts of both groups clinched the interpretation. Adult patients, dying from tuberculosis during the war, had a very severe degree of damage to the arteries of their hearts. In fact, had not tuberculosis won the race, the identical individual would have died from a heart attack in a very short time. The conditions of war had altered his resistance to tuberculosis and moved his death from the statistics of heart attacks to that of tuberculosis. Diet had nothing to do with it.

Two years later the conditions were reversed. The antibiotics against tuberculosis had become available, and deaths from this disease fell like a lead pipe. Immediately deaths from heart attacks started to rise. The autopsies gave us the answer; the adult dying from a heart attack had healing tuberculosis in his lungs. The antibiotics had stopped immediate death from tuberculosis giving the advanced arterial damage in the heart a chance to become the winner. This is a very simple explanation for the modern rise in heart attacks. It fits all the facts and does not involve theoretical changes in the environment and diet. These may have a minor effect, but they are unimportant when compared to the tremendous number of lives saved from tuberculosis. The details of this investigation were published in 1974.[1] It was pointed out that the conversion of tuberculosis sanitariums to general hospitals is timely; the institutions are now caring for heart attacks. Twenty-five years ago, the identical individual might have entered with tuberculosis.

This is not the first association of tuberculosis and atherosclerosis (hardening of the arteries). As early as 1919, Herxheimer[2] noted in the routine autopsies in Munich that patients dying with tuberculosis had an advanced degree of arterial damage. I confirmed his observations in reviewing the autopsy protocols at Graz; it was unusual to find macroscopic (visible to the unaided eye) damage to the arteries of those below the age of 30. However, when such a case did occur, it was usually in a

person who had died of tuberculosis. In the adults having died from tuberculosis, advanced atherosclerosis was the rule.

The development of the science of Pathology gave new tools for the early diagnosis of atherosclerosis. Cutting tissues into thin slices, staining with appropriate dyes, and studying these preparations under the microscope permitted detection of damage before it would be obvious at a routine autopsy. In 1925, Zinserling[3] studied the arteries of a series of children who had died from infectious diseases. He noted that in some, beginning atherosclerosis was apparent as early as six months of age. In others, it was not present until the age of four, but after that time, it was the rule to find detectable atherosclerosis. Strong and a team of investigators[4] in 1969 confirmed Zinserling's observation in a study of tissues from the Charity Hospital in New Orleans, Central America, South America, and in South Africa. All of the children in this large group had atherosclerosis after three years of age. Since most children die from infectious diseases, it would appear that the individual susceptible to any infectious disease is highly vulnerable to atherosclerosis. Tuberculosis does not have a corner on the market.

There seems no doubt from the last two references that children dying from infectious diseases have started the process leading to heart attacks. *It is apparent that the conquest of tuberculosis, pneumonia, meningitis, septicemia (infection in the blood) and scores of other devastating infections by the antibiotics has been the major factor in the explosive rise of heart attacks.* The number of individuals surviving beyond middle age has mushroomed; consequently, a NEW POPULATION[5] of adults is alive for the first time in history.

The significance of Zinserling's observations escaped detection for many years because heart attacks were rarely seen at the time. Yet they laid the foundation for a clear understanding of why heart attacks have become so numerous. During all of recorded history, part of the population has survived into old age. Heart attacks were so rare among them that the disease was not established until the 20th century. These individuals were relatively immune to arterial degeneration. The other segment of the population died comparatively early from infectious diseases. As soon as those susceptible to infections began to live beyond middle age, heart attacks exploded. Zinserling found that this section of society began forming atherosclerosis in their arteries by the age of six months. Individuals susceptible to infection have no immunity

against heart attacks but are highly vulnerable. This is a NEW POPULATION appearing on the scene during the 20th century.

In nature there is a simple explanation for each occurrence although the answer is frequently elusive. Sometimes the answer is as obvious as the nose on your face, but we fail to see it. The physiology explaining the association of infections and hardening of the arteries has been around for a long time, but the forest has been obscured by the trees. It will be learned in Chapter 3 that one of the most potent factors in heart attacks is a thyroid deficiency. This was clearly established in the last century, but it was forgotten since there were no heart attacks around at the time. The details will be revealed later; only a few salient facts will be mentioned here.

Thyroidectomy (removal of the thyroid gland) in animals or in the human soon leads to atherosclerosis. Thyroid administration will delay or prevent this degenerative process. It was firmly established in the last century that the thyroid-deficient animal or human was unusually susceptible to infectious diseases. Sherlock Holmes would not have missed these two interlocking clues. If deaths from infection were stopped, one might expect that the process of atherosclerosis would continue until "death do us part." It has happened. The details of how it is accomplished are also quite simple. In 1877, Ord[6] demonstrated that in the tissues of thyroid-deficient patients a glue-like substance, called mucin, held on to water and started degeneration. In 1955, Andersen[7] found that this same glue-like material developed in the tissues of children born without sufficient thyroid. The tissue content was normal as long as thyroid was administered, but within six weeks, if thyroid therapy were stopped, mucin rose rapidly. If thyroid therapy were started again, the mucin content returned to normal.

The story now seems complete. Heart attacks were rare as long as infectious diseases killed off the susceptible individuals at an early age. *In previous centuries over half of the population never reached middle age, and consequently had no opportunity of dying from heart attacks.* Better medical care and the antibiotics during the 20th century have stopped the carnage from premature deaths due to infections. It has now been demonstrated that those susceptible to infections are also more vulnerable to heart attacks. This NEW POPULATION is the major factor in the explosive rise of heart attacks in the 20th century. Does this represent a deterioration in the state of the health of the

world? Quite to the contrary. Many more years of fruitful living have been added.

This improvement can be well demonstrated by my studies[1] on the routine autopsies at Graz, Austria, which were mentioned above and which were reported in 1974. Between 1930 and 1970 heart attacks rose 1000 percent, far more than any other disease. This cause of death was replacing only deaths from infections as no other diseases showed any decrease. Heart attacks were not occurring at an earlier age since the average age of death from this disease in 1930 was 68 years and in 1970 was 66 years; this difference is not statistically significant. Furthermore, in 1970 with the tremendous rise in heart attacks only 6 percent of them occurred before the age of 50 years. The senior citizens are responsible for most of them with 38 percent appearing after the age of 70 years.

The tremendous improvement in health, in spite of the rise in heart attacks, is seen from the average age of survival. In 1930 only 47 percent of all deaths occurred above the age of 50 years; in 1970 this had risen to 67 percent. It is obvious that heart attacks signify that health has been benefited to the point that patients now live long enough to contract the disease rather than dying at a much earlier age from infections. Our problem is not how to stop heart attacks, per se, but how to prevent the minority of them occurring at an early age. There is a physiological difference between the patient who dies with coronary occlusion (interruption of blood flow to the heart) at 23 years of age — the lowest in my experience — and another at age 80.

The following chapters offer a rational approach to the prevention of early heart attacks.

CHAPTER II

What is a Heart Attack? How Often is the Diagnosis Wrong?

The heart, situated behind the left breast, is the most important piece of meat in the human body. Its sole purpose is not for love but for carrying fresh food to each of the billions of cells in the body and for carrying away garbage. In other words, it is a pump that moves blood continuously through the blood vessels — much like a river carrying commerce to various cities.

Anatomically the heart is a complex bundle of muscles and nerves containing four cavities which are equipped with valves that insure the flow of blood in the proper direction. The muscles on the right side of the heart are not as thick as those on the left since the right heart forces blood only through the lungs and again back into the left side of the heart. The left side of the heart must move the same quantity of blood not only through the head on one end but also the feet on the other end and finally back into the right side of the heart. The more powerful left side of the heart must have a greater blood supply for its muscles.

Leading from the left side of the heart is a large tube called the "aorta" whose branches must carry blood to the far corners of the individual. The first branches to leave the aorta are the coronary arteries which supply the muscles of the heart. They run over the external surface of the heart in small grooves so that they are not compressed when the heart muscle contracts. *Any pathological process interfering with the blood flowing through the coronary arteries may cause a heart attack.*

In a broad sense, any means of interfering with proper heart contraction may produce sudden death. An electrical impulse spreads through the heart causing the muscles to contract in an orderly manner. An electrical shock such as from lightning or from an electrical current may interfere with the propagation of the electrical impulses in the

heart in which case a heart attack would occur at once. Such deaths seldom happen and are usually classed as "shock" rather than "cardiac."

The usual heart attack — the one that is feared — is due to the hardening of the arteries or atherosclerosis. This is an insidious process that may have been going on for years before the restriction of blood flow causes any symptoms such as chest pain or shortness of breath. Rarely a tiny clot may break off elsewhere in the body, lodge in a coronary artery, and lead to instant death without any pain. There may be no damage to the structure of the coronary artery in this instance. Only an autopsy will disclose the true cause of death in these rarely-seen cases; they are termed "coronary embolisms."

The coronary arteries may also be occluded suddenly if a blood clot forms locally in these particular arteries. This happens frequently in arteries containing considerable atherosclerosis; the clot is only the end point. Often the degree of hardening of the artery is mild and has not caused any chest pain. These clots, superimposed on the atherosclerosis, may occur at night when the circulation is diminished. The individual may die suddenly without being awakened by the clot.

The usual course of events, however, is for the patient during exertion to experience chest pain which disappears in a few minutes if one lies down. This limitation in activity increases as time elapses; eventually vigorous activity must be abandoned. Medications that dilate the arteries will give some temporary relief, but they do not eliminate the cause of the problem. As the artery carrying blood to the heart progressively narrows due to atherosclerosis, the heart screams with pain from a lack of circulation, and effective muscular contraction is lost. Death soon ends the misery.

Unfortunately sudden death has become synonymous with heart attack. No doubt heart attacks are numerous, but many other diseases may cause sudden death. The death certificate must carry a diagnosis, and "heart attack" is convenient. A discussion of other causes of death will follow, but two instances of my acquaintance will illustrate the point.

A 51-year-old male was employed by a Building and Loan Association. He had retired from 20 years of service in the Air Force and had always passed his military examinations without evidence of disease. For 12 years he had been a patient of mine and was on thyroid therapy. Annual examinations included a chest x-ray, electrocardiogram, urinalysis, many blood tests, as well as the interim history and the physical examination. His last examination was made May 28, and all

values were well within the normal range. In September he collapsed at work while enroute from one office to another. There had been no warning or evidence of serious disease. The coroner's verdict would have read, "heart attack," had not an autopsy been performed. The post-mortem revealed a normal heart. Sudden, painless death was due to rupture in the brain of an artery that had been dilated since birth.

The second was a surgeon in his late sixties who had had an elevated cholesterol for some years and in whom one might have expected hardening of the arteries. While playing golf — his favorite pastime — he suddenly collapsed with extreme weakness. He died in the ambulance enroute to the hospital. Another heart attack? No. The autopsy revealed that the large artery, the aorta, which carries blood from the heart to the abdomen had ruptured; consequently he had bled to death internally. Again only the autopsy prevented another wrong diagnosis.

These two cases illustrate clearly that sudden, unexplained death frequently may be due to a cause other than a heart attack; without an autopsy the clinical diagnosis is often a very poor guess. No one knows how often such mistakes are made, but the following facts, illuminated by two series of autopsy studies, indicate that among sudden death, heart attacks are in the minority.

WHAT ISN'T A HEART ATTACK

Any sudden, unexplained death not due to occlusion of one or more coronary arteries is not a heart attack. Yet in the absence of an autopsy, such a death is usually signed out as a "heart attack" by the family physician or by the coroner, in case the patient was not seen by a doctor recently. Granted that more of these individuals die from coronary disease than from any other *single* disease, still the total of non-coronary, sudden deaths exceeds the total of heart attacks.

One seldom hears about the deaths from the multitude of other causes since the only way they are recognized is from the autopsy. Rarely are postmortem examinations made due to public apathy. Experience in Austria, where autopsies are mandatory, clearly indicates that, once established, the public thinks no more of a routine autopsy than of a tonsillectomy or excision of the gallblader. As a matter of fact, the autopsy is far superior to surgery since the patient does not awaken with the confusion, vomiting, and headache so often encountered after an operation.

The renowned Empress Maria Theresia invited the Dutch physician, Gerhard van Swieten, to come to Austria in 1745. He became physician-in-waiting for the Empress, taught at the medical school, and held directorship of the court library. The public health of Austria was in a horrible condition at the time. Van Swieten pointed out that at the municipal hospital, out of 600 consecutive births, 580 children perished. A law was passed that each death in a hospital should have an autopsy — an edict that still stands today.

Austria became the Mecca for medical education. They no longer guessed at the cause of death, but in each case the physician could see any mistakes he had made. So many of the American physicians flocked to Vienna for post-graduate study that a group of them founded the American Medical Society of Vienna in 1879. This organization is still in existence and arranges courses for itinerant physicians visiting the home of music and the waltz. It was through the American Medical Society of Vienna that I became acquainted with the invaluable records of routine autopsies which solved the riddle of heart attacks. This will be discussed in a later chapter.

If the American physicians returning from Vienna for the past 100 years had insisted on routine autopsies in America, the *Riddle of Heart Attacks* would not be news today, and countless millions of dollars as well as precious time of many scientists would have been spared. Little did Maria Theresia realize two hundred years ago that her proclamation would solve the major problem in medicine in the 20th century.

Although most deaths in the United States are not autopsied, it is only from the infrequent ones that the true incidence of heart attacks can be assessed. The abuse of calling each sudden, unexplained death a heart attack was the subject of an editorial in one of our leading medical journals (*New England Journal of Medicine,* 262:149, 1960). Entitled "The Convenient Coronary," the article discusses a review of 3557 death certificates in Pennsylvania where the error of diagnosis was estimated to be between 27 and 63 percent. Such errors are hardly an educated guess. Yet, it is from these inaccurate statistics that money is raised for research, and the figures are used to convince the public that the world is coming to an end.

Only two sets of autopsy data will be mentioned here to refute the claims that heart disease must be stopped at all costs. The first comes from autopsies of military personnel during World War II. There

were more than 40,000 autopsies studied between January 1942 and January 1946. Those under 40 years of age, having died unexpectedly, were studied by Mortiz and Zamcheck (*Archives of Pathology* 42:459, 1946). Here was a group of apparently healthy men who had passed physical examinations to enter service, and were under close medical supervision. They had available free medical care including hospitalization, the Utopia so strongly urged us by some politicians of the moment. None of them were incapacitated for more than 24 hours, or they would have been forced on sick call by their superiors.

There were approximately 1000 cases between the ages of 18 and 40. Were these sudden deaths all heart attacks? In civilian life they would have been so labeled unless an autopsy had been performed. In reality, less than one-third were due to heart attacks. It is apparent that the significance of heart attacks below the age of 40 has been, as Mark Twain said about the reports of his death, "grossly exaggerated."

Space allows only time to mention two other conditions causing sudden, unexplained deaths in young soldiers. Ten percent of the total were due to hemorrhage in the brain. Even socialized medicine can do nothing toward the detection nor the prevention of these untimely deaths. Rupture of the blood vessels do not depend upon exertion since one-third occurred during sleep and only 17 percent during exercise.

Another 10 percent of the young adults suffering sudden death was caused by meningitis. Death usually occurred within 24 hours after onset of symptoms, and 7 of the 110 deaths in this group were dead in the barracks without having been on sick call. About 15 percent of the young, sudden deaths had nothing to explain their deaths even at autopsy, but heart attacks could be definitely ruled out. Hence, it is apparent that to label all sudden deaths "heart attacks" is as serious an error in mathematics as a drunken husband's alibi.

The second report[8] clearly indicating that heart attacks are in the minority of sudden deaths comes from a series of 275 consecutive autopsies among ages between 20 and 45 seen in New York City by the coroner. Only 28 percent were due to coronary artery disease, less than one-third of the total.

Deaths from respiratory diseases accounted for 18 percent and one-seventh of these were due to tuberculosis. This disease cannot be written off as unimportant in spite of antibiotics. Pneumonia, asthma, and other respiratory diseases completed the picture. Deaths from dis-

eases of the central nervous system accounted for 22 percent of the total. Hemorrhage and meningitis accounted for most of them just as in the soldiers in World War II. The gastrointestinal tract made up 13 percent of these untimely deaths, while 4 percent were due to diseases of the urinary tract with kidney infections predominating.

These two excellent autopsy studies clearly indicate that more than two-thirds of the sudden deaths, usually listed as heart attacks, are due to diseases that may arise anywhere in the body. The phobia against heart disease must stop, and a more rational explanation for our changing death patterns must be found. Certainly more medical care is not going to solve the problem, since the military with unlimited medical facilities has about the same ratio of sudden deaths as is seen in civilian life.

If the actual number of heart attacks are less than one-third the number recorded, are they the menace that they are alleged to be? The answer is an emphatic, "NO." The misleading figures are being used by those investigators so anxious to feather their nests from more grants from the government or from the donations to health agencies that support research. By keeping the exaggerated problem before the public, Congress is spurred for larger sums to be appropriated for investigation. This seems to be an unjustified waste of public funds.

A practical method of eliminating premature heart attacks is presented in the following chapters.

CHAPTER III

The Cause of Heart Attacks

Credit for realizing that thyroid deficiency is one of the most potent factors in causing heart attacks must go to the pathologists of Vienna about 1890. They did not appreciate the significance of their discovery since heart attacks had not been described as yet; they could hardly claim credit for curing a disease that did not exist. However, their keen observations led to some experimental work in which atherosclerosis was produced in animals, again long before the new disease in man was described. For 85 years conclusive evidence has been accumulating to substantiate their observations, but we must go back even further to understand how the pathologists happened to make their outstanding contribution.

Our story begins in 1873 when Sir William Gull[9] discussed before the Clinical Society of London five peculiar cases in women. These patients had many of the features of cretinism, a condition known to be associated with a lack of thyroid activity. Cretin children fail to grow and develop normally either physically or mentally. In Gull's adults the condition appeared long after growth and development were completed; otherwise, there was similarity in appearance and behavior.

Another astute London clinician, Dr. William M. Ord,[6] was fortunate enough to obtain an autopsy in 1877 on a similar case in whom he had watched the disease progress for six years. There were no unusual diseases in her family. She had borne two normal children, went through the menopause at age 44 and had considered herself well until the age of 49 when she began to have "shivering spells" and on occasions would pass bloody urine. Previously she had worked hard as a seamstress to support her ailing husband. Soon her hands became numb and clumsy; to handle a needle was difficult. Her mental faculties decreased; her movements were slow and awkward; she had difficulty in speaking, and she fell asleep unless she was moving about. Her efficiency decreased to the point that it took her two hours to dress. Her skin and

especially the face became swollen until there was no expression. She died at the age of 60 in a coma.

At the autopsy there were several outstanding findings, the most remarkable of which was a swelling of the skin and of all of the connective tissues. Apparently, the fluid was held in combination with something such that the cut surface did not release water. This was quite different from cases of kidney failure in whom fluid would run freely from any incision. Other findings relative to our present discussion were: (1) the thyroid gland was replaced with fibrous tissue, and (2) the entire arterial system showed advanced atherosclerosis. The arteries to the kidneys, to the brain, as well as the coronary arteries to the heart were lined with deposits limiting the flow of blood. Ord decided that the absence of the thyroid gland was responsible for these pathological changes.

He called in an expert chemist to determine the nature of the substance that was binding the water in the tissues. This was a new phenomenon that had not been encountered in the past. Chemical analysis revealed that those tissues with a high water content contained an unusual amount of mucin, a thick, glue-like material. This suggested a name for the new disease, and Ord coined the term "Myxedema," "Myx" — coming from the Greek word meaning mucin, and "edema" — describing the water-logged tissues. This name has persisted to the present day and signifies a marked degree of thyroid deficiency. Still, there are patients with subnormal thyroid function in whom the tissue-swelling is not so prominent.

At once, other physicians recognized similar cases, and realized that this new clinical entity was rather frequent. So much interest was aroused that in 1883 the London Clinical Society created the first "Think-tank" in medicine to thoroughly study the new disease. Some of the leading clinicians, a chemist and a physiologist made up the 13 member Commission. For five years they collected similar cases, corresponded with investigators in foreign countries, and tabulated information totaling over 300 pages in the published report of 1888.[10]

Over 100 cases of myxedema had been collected. The chemist had confirmed that mucin was the agent holding on to water. Sir Victor Horsley, the physiologist, had removed the thyroid gland from the monkey, swine, sheep and donkey. In each instance within a matter of a few weeks, the experimental animals developed swellings and an excess of the mucin in their connective tissues. The clinical syndrome

had been produced in the animals by removing the thyroid, leaving no doubt about the cause of myxedema. Consistently, autopsies on humans confirmed a decreased function of the thyroid gland.

About the same time, Professor Kocher, a surgeon in Berne, Switzerland, was making some similar observations. For centuries, in some regions of the world, the thyroid gland would enlarge so much that the windpipe would be compressed, and the patient would suffocate. These growths were called goiters and their regions of occurrence were called "goiter regions." Kocher[11] perfected a technique whereby the entire thyroid could be removed with a reasonable chance of saving a life, and in 1883, reported 101 cases. Not long after removal of the entire thyroid, symptoms similar to those described by Ord in 1877 began to develop. Kocher called this miserable condition "cachexia strumipriva," or debility due to loss of the thyroid. He had prevented strangulation only to have the patient die from myxedema in a few years. He soon modified his operation and left some of the thyroid intact; this practice is still used today.

Professor Billroth (the eminent Vienna surgeon who perfected the stomach-ulcer-operations that are still used today), was also seeing some of the huge goiters. His experience was the same as Kocher: after total thyroidectomy, the patients soon died from myxedema. Austria was in a unique position to make a major contribution; in compliance with the law, all hospital deaths were autopsied. The pathologists soon noted that after total thyroidectomy autopsies revealed an exaggerated hardening of the arteries. This confirmed Ord's autopsy of advanced atherscleroses in his original case of 1877.

Professor Billroth was a scientist as well as a skillful surgeon. The marked damage to the arteries aroused his curiosity. He assigned one of his students to investigate the arterial changes in animals after removal of the thyroid gland. The report by von Eilsberg[12] in 1895 clearly showed that removal of the thyroid from the sheep or goat would produce arterial degeneration in arteries throughout the animal including the coronary vessels supplying the heart. It was fortunate that herbivora — animals that eat only plants — were chosen for the experiment since these animals never eat cholesterol-containing foods. Cholesterol is not found in the plant world. This was long before cholesterol was associated with atherosclerosis. This early experiment clearly demonstrated that thyroid deficiency, and not dietary choles-

terol, was implicated in arterial degeneration whose characteristics were similar to those in the human.

By this time active preparations were available for treating thyroid deficiency. In 1891 Murray[13] found that a glycerine extract of fresh animal thyroids could be injected into humans suffering from myxedema, with complete relief of the symptoms. In fact, the first patient whom Murray treated began medication in 1891 and stopped treatment in 1919 when she died at the ripe old age of 72. She had been shifted to oral tablets after it was found that the hormone was not destroyed by digestion. However, her life had been saved by the glycerine extracts started 28 years earlier.

A few years after von Eilsberg had produced arterial degeneration in the herbivora by thyroidectomy, Pick and Pineless,[14] also in Vienna, repeated his work and carried it one step further. Not only were they able to confirm the degeneration in the arteries going to the heart of goats, but if they gave thyroid hormone to the thyroidectomized animals, they could prevent the atherosclerosis. Then the story was complete. A disease of man could be produced in the experimental animal merely by removing the thyroid gland, and the administration of thyroid hormone could prevent the disease in the susceptible animal. Thus, before heart attacks were described as a clinical entity, their cause and cure were known. However, there was no place to use this knowledge, and it was forgotten by the time it was needed.

In 1913 a Russian physiologist, Anitschkow[15] reported an experiment on rabbits that was to change the history of atherosclerosis for 60 years. He fed huge doses of cholesterol to the rabbit, a vegetarian, that never eats cholesterol-containing foods and whose liver is not equipped to excrete the excess. The concentration in the blood went up several hundred percent; such concentrations proved toxic. Stress caused some atherosclerosis of the blood vessels. This was not a physiological experiment, but a pathological finding. Anitschkow realized that it had no practical significance, and never suggested the use of diet in controlling atherosclerosis.

Similar administration of cholesterol to the rat, the dog, or even the human is not followed by such elevations in serum (blood) cholesterol or atherosclerosis in the arteries. These species ingest cholesterol in their diets, and their livers possess enzymes that destroy the excess cholesterol. Although it will be pointed out in the final chapter that cholesterol is not important in atherosclerosis, it seems timely that from

Anitschkow's laboratory in 1965 there came an explanation for the observations on rabbits. Malasheva[16] reported that huge doses of cholesterol suppressed the thyroid function in rabbits as completely as thyroidectomy. As pointed out above, any means of reducing thyroid activity leads to atherosclerosis. The atherosclerosis in the cholesterol-fed rabbits was due to the low thyroid function and not due to the cholesterol itself.

Even war sometimes makes a contribution to science. It was during World War I that the next great stride was made in establishing thyroid deficiency as the culprit in atherosclerosis. Zondek,[17] a German physician, noted that some cardiac cripples displayed many of the signs and symptoms of myxedema. Such cases failed to respond to the usual therapy of digitalis (a heart medication). Their hearts were enlarged, their tissues were water-logged, they were short of breath, they were weak, and some were bed-ridden. To his amazement, thyroid therapy soon restored their health and they were back to work, or if in the military, they returned to duty. He named the new entity "Myxedema Heart;" it differed in no way from myxedema described above except that the heart failure was predominant.

Following the war, Zondek wrote a book, *Diseases of the Endocrine Glands,* published by Williams and Wilkins, Baltimore, Md. This was translated into English in 1944. Every physician should read this masterpiece. In the chapter on Myxedema, page 180, he shows electrocardiograms displaying the low voltage as a result of the feeble beat of the heart. Following thyroid therapy, when the heart is contracting in the normal manner with more power, the voltage on the electrocardiogram (EKG) is returned to normal excursion. I have found that often the low voltage on the EKG reflects the status of the thyroid better than the commonly used blood tests.

In 1919, another German physician, Assmann,[18] confirmed Zondek's observations on Myxedema Heart and pointed out that no age is exempt. His 20-year-old lieutenant had to be evacuated from the front lines because of a loss of memory, extreme fatigue, and inability to carry on his duties. The heart was markedly enlarged, there was edema of the skin and extremities, the temperature was quite low, the pulse was 50 and the blood pressure was low. There was no response to digitalis and diuretics (medication to help eliminate excess water from the body), but following Zondek's lead, thyroid therapy promptly elevated the pulse, the blood pressure, and the energy. The heart returned to

normal size, and the other symptoms all disappeared. *It is obvious that young individuals can develop severe, incapacitating heart disease as a result of thyroid deficiency and that this disease is readily amenable to thyroid therapy.*

Myxedema Heart came into focus all over the world. Scores of reports soon appeared; only a few will be mentioned. Several important lessons were soon learned, but some physicians were negligent in keeping up with the new information. Christian[19] in Boston had one patient who died suddenly before any medication was given. An autopsy revealed marked narrowing of the coronary arteries so typical of heart attacks. The same author had another patient, a 73-year-old woman, who had been taking thyroid for 25 years. She stopped the medication because she felt it was causing a pain in her shoulder. Soon she developed angina (chest pain) and other symptoms of an impending heart attack. After returning to small doses of thyroid, again her symptoms disappeared.

For 10 years, hundreds of patients with evidence of heart disease were improved by small doses of thyroid. It was soon learned that the thyroid could not be used recklessly for heart failure, as digitalis had been in the past. It was customary to give large doses of digitalis, and then curtail the dosage as improvement began. But, the large doses of thyroid put too much of a load on the heart, and death might occur in a short time. On the contrary, a small dose of thyroid in the beginning, followed by a gradual rise in dosage, lead to a marked improvement in the angina, in the size of the heart, and in the condition of the patient. In 1925 Sturgis[20] reported in the *Journal of the American Medical Association* 26 well-studied cases from Harvard. For individuals with heart disease only one grain of thyroid daily was recommended in the beginning and usually not more than 2 grains were necessary for maintenance.

As time passed, it became apparent that heart attacks were more than a disturbance in the cholesterol in the serum. It seems ironic that Anitschkow should be frequently quoted for his cholesterol-feeding to rabbits which he admitted at once had no significance, yet rarely does one find any reference to a major contribution from his laboratory by one of his students, Friedland.[21] The latter began working on cholesterol and other fats in 1927 and published his full report in 1933. He found that if he fed the rabbits large quantities of cholesterol, as Anitschkow had done, he could prevent the high serum cholesterol

levels and the atherosclerosis by simultaneously giving some thyroid hormone. He reasoned that the thyroid was controlling the other fats in the blood as well as the cholesterol. Any of the fats might be affecting the atherosclerosis. He even advocated the use of thyroid prophylactically in the human for those with an elevation of the serum fats. If this contribution had been pursued, atherosclerosis would have been conquered years earlier and much time would have been saved.

In addition, Simonds brought out the close association of the thyroid with fat metabolism in 1932.[22] He found that making dogs *hyper*thyroid (too much thyroid hormone) with administration of extra thyroid, the animals could tolerate much larger quantities of fat without elevating the level of fats in the blood. If the fat levels in the blood are elevated in *hypo*thyroidism (too little thyroid hormone), it is no surprise that they should be found low in *hyper*thyroidism since the elevated metabolism would burn up more fat.

Hurxthal, a physician at the Lahey Clinic, where many hyperthyroid patients were operated, was in an excellent location for the study of this question. In 1934 he found that the patients with too much thyroid secretion had serum cholesterol levels below the normal range.[23] After the thyroidectomy, the cholesterol would start to rise, and if too much thyroid had been removed, the cholesterol would rise above the normal range. Under these circumstances administration of some thyroid hormone would bring the cholesterol down to the normal level. The relation of cholesterol to the metabolic rate was so sensitive that Hurxthal suggested the use of the serum cholesterol as a diagnostic test for thyroid function. He suggested that an elevated serum cholesterol was an indication for thyroid therapy, if no other reason could be found for the elevation.

Hurxthal's observations were on adults and in them the cholesterol does often reflect the thyroid state. I have routinely run cholesterols on all patients during the past 25 years. Those patients with elevations usually need thyroid therapy, but some adults and most younger patients may run normal or below normal cholesterol readings, yet be in dire need of thyroid. Two conditions, both rare and easily ruled out, may elevate cholesterols and still do not need thyroid. One is nephrosis, a condition in which large quantities of protein are lost in the urine. Since much of this protein must be manufactured in the liver, this may interfere with the elimination of cholesterol by that organ. Liver damage may also be associated with an elevated cholesterol since the excre-

tory process is retarded. But, these two exceptions do not interfere with the routine use of thyroid for hypercholesterolemia (too much cholesterol in the blood).

Some additional information on feeding rabbits cholesterol was added by Turner in 1938.[24] He repeated Anitschkow's work of 1913 as well as Friedland's in 1927. Indeed, some strains of rabbits developed a high serum cholesterol and atherosclerosis after prolonged administration of cholesterol, while other strains, fed in the same manner, developed neither the elevation in serum cholesterol nor the damage to the arteries. He found that the response was related to the function of the thyroid. If he administered thyroid along with the cholesterol to the strains developing arterial damage, they were protected from atherosclerosis just as Friedland had reported. These results suggested that it was the thyroid deficiency which was responsible for the damage and not the cholesterol itself. Proof was found by thyroidectomizing the strains of rabbits in which cholesterol feeding produced no deleterious effects. These animals then lost their immunity and developed both elevated cholesterol levels in the serum and atherosclerosis in their arteries.

Thus, years before the rapid rise in heart attacks, cholesterol was exonerated as the culprit, and thyroid deficiency was firmly established as the cause of atherosclerosis. Another much neglected report was by Gildea[25] from Yale University in 1939. He and his colleagues were studying a series of patients with elevations in the serum fats. Of the fatty acids in the blood, 50 percent is united with phosphorus and protein, the so-called lipo-proteins, 25 percent is combined in triglycerides, 20 percent is united with cholesterol and only 5 percent is free fatty acids. Some investigators have felt that the elevated lipo-proteins are responsible for atherosclerosis rather than cholesterol. Others have blamed the triglycerides alone. Gildea found that all of the serum fats could be brought down to normal levels with the use of nothing but thyroid therapy.

Some of the patients in his series were definitely hypothyroid with the usual symptoms and low metabolic rates. Others had neither the symptoms nor the low metabolism, but both groups responded to thyroid therapy with a lowering of the fats in the blood. In retrospect, it seems plausible that most of the patients had some degree of thyroid deficiency since the basal metabolism test for thyroid function is not fool-proof. At any rate, it was clearly demonstrated that elevated serum

fats would respond to thyroid therapy in small dosages without any evidence of harmful effects.

During the 20-year-interval since Zondek had described Myxedema Heart, hundreds of patients were successfully treated with thyroid hormone. It became obvious during this interval that the new disease, heart attack, was becoming much more frequent, and autopsies left no doubt that they were due to atherosclerosis of the coronary arteries so intimately associated with thyroid deficiency in either experimental animals or spontaneously in man. Many patients with the symptoms and physical findings suggesting an impending heart attack regained their health on small doses of thyroid.

Then came a terrorist's bomb. In 1938[26] a recently graduated physician, lacking in experience and apparently unacquainted with the enormous literature establishing thyroid as a safe medication in Myxedema Heart, wrote a scathing attack on the use of thyroid in heart disease. His single experience was in a female 63 years of age who had been diagnosed hypothyroid five years previously and had been treated successfully with thyroid by another physician. She had not received any thyroid for two years when she consulted the young physician for anginal pain requiring nitroglycerine (which dilates arteries) for relief. The basal metabolism was minus 41 percent and the serum cholesterol was 540 mgs leaving no doubt that she had Myxedema Heart. She was put on *4 grains* of thyroid daily and died with a heart attack on the 8th day.

Obviously this physician was not acquainted with scores of reports over the previous 20 years nor with the warning of Sturgis in 1925 that such patients with heart disease should be started on one grain daily and seldom needed more than 2 grains for maintenance. The recent graduate then reviewed the literature and emphasized that eight deaths had been recorded previously. These were the cases that had occasioned the previous warnings for the use of a small dosage. I reviewed each of these reports and found that from *4 to 30 grains* daily had been used in the fatal cases. Such colossal errors in therapeutics would lead to disaster with any medication. The neophyte investigator made no mention of the hundreds of cases treated successfully with the proper dosage of thyroid, but only emphasized the "potential danger" of such therapy.

Years of progress were lost because few physicians had the opportunity to review the extensive literature, and only took the latest report

as "gospel" on the subject. To this day many physicians, nurses and health educators remark that thyroid therapy is likely to cause heart disease and would not think of using it as a prophylactic procedure.

It was probably this tragic publication that caused a very seasoned investigator to err in the opposite direction. In 1946 the late Dr. Paul Dudley White and Jakob Lerman[27] made a series of metabolic studies on 28 patients under the age of 40 recovering from heart attacks. The majority showed low metabolic rates and high serum cholesterols ranging up to 490 mgs. Thyroid therapy was tried with gratifying results; no angina was precipitated in those free of this complaint; angina present at the start disappeared in all but two cases; the basal metabolic rate tended to go up, and the cholesterols fell.

These investigators were on the threshold of a successful preventive and prophylactic program for heart attacks, but their observations were not pursued. Years later when my own work indicated identical findings I wrote to Dr. White to inquire why he had abandoned the use of thyroid. He replied that after a few months the cholesterol levels began to rise again, and he had felt that this was not the answer. The last line in his letter revealed the unfortunate mistake. He said that perhaps their dosage was too small for they had been using only one-tenth to one-quarter grains daily. The scare in 1938 may well have influenced their judgement, or I would not be writing this book in an attempt to straighten out the confusion. If White and Lerman had used the previously recommended one grain daily, the riddle of heart attacks undoubtedly would have been solved many years earlier. It was Dr. White who was called from Boston when President Eisenhower had his heart attack in Denver.

In 1951 a milestone was passed. Professor William B. Kountz[28] of Washington University in St. Louis decided that the physiology pointed clearly to thyroid deficiency as the culprit in atherosclerosis. In a monograph entitled, *Thyroid Function and its Possible Role in Vascular Degeneration,* he reviewed the literature up to that time as mentioned in Chapter One, and did not fail to use small doses of thyroid as suggested by Sturgis in 1925. He collected 288 patients with low basal metabolic rates, many of whom had elevated serum cholesterol levels.

The patients were separated into three age groups, the first being business men, with an average age of 55 years, who were private patients in his practice. Many of them had very little evidence of atherosclerosis at the start of the study. The second group were out-patients at the

University Clinic, with an average age of 61 years. Many in this group had evidence of atherosclerotic damage to the heart and other organs. The third group was still older and averaged 67 years of age. They were confined to an infirmary and presented advanced generalized atherosclerosis, some even having the arteries in their legs showing calcium deposits on the x-ray.

Some of each group served as controls while others were put on small doses of thyroid over the following five years. The number of heart attacks or strokes were recorded for each age group and for both the controls and the treated subjects. Among the younger business men there were no deaths among the thyroid treated subjects, but among the controls 15 percent had fatal strokes or heart attacks. Obviously, the patients with low thyroid function were susceptible to atherosclerosis, and if thyroid therapy were started by age 55, many premature deaths could be prevented.

In the patients with demonstrable moderate atherosclerosis averaging 61 years of age, the results of thyroid therapy were again dramatic. Among those receiving thyroid therapy, a 3 percent mortality occurred. In contrast, the controls suffered a 19 percent loss — over 600 percent greater death rate. Even in the advanced-age and arterial-damaged group, thyroid therapy again demonstrated its efficacy. At the end of the observation period these patients had an average age of 72 years, yet there were one-half as many deaths among those receiving thyroid as among the controls. These clear-cut results should have started the investigators back on the use of thyroid. This was the first controlled study reported. Furthermore, there were no dangers from thyroid therapy among this large group of patients. The errors in dosage, which had caused the scare in 1938, were avoided.

However, by this time many of the scientists and clinicians were off on the cholesterol tangent. Huge sums were being appropriated for research in this field; the money must be spent so more would follow. There was no time to consider any other approach since the theorists promised that all that was needed was to lower the cholesterol levels in the blood, and the food manufacturers guaranteed that it could be done with polyunsaturated fats. This mammoth error will be unmasked in the last chapter. It became obvious as time passed that heart attacks were climbing year after year in spite of modifying living habits and diets. Obviously there was an error some place, but the

cholesterol enthusiasts have a professional standing to protect, and they are going to fight to the bitter end.

The final proof that the thyroid deficiency was the missing link in atherosclerosis came from Denmark in 1955. The mucin, causing the edema in Ord's original autopsy in 1877 and promptly appearing in Sir Victor Horsley's thyroidectomized animals in 1888, has received a tremendous amount of investigation in the meantime. It has been found that there are many compounds having the property of holding onto water; they have been renamed mucopolysaccharides. They are always present in atherosclerosis and in most other pathological states such as cancer, arthritis, hypertension, diabetes, etc. They are combinations of complex proteins with one of the sugars in the molecule. It is probably more than coincidence that their concentration in the connective tissues promptly increases in thyroid deficiency. Andersen,[29] using a tiny punch-biopsy of the skin on the arm, studied the mucopolysaccharide content in 99 children between the ages of three months and 15 years. Among the group were 52 children with no evidence of thyroid deficiency; none of them showed any elevation of the mucopolysaccharides in the skin. There were 26 with symptoms of thyroid deficiency and were on therapy. If the thyroid were stopped for a month, the skins began to show an abnormal accumulation of the mucopolysaccharides. If the youngsters were again put on thyroid therapy, the concentration of the deleterious mucins declined. Andersen suggested analysis of the skin for mucopolysaccharides as a superior method for diagnosing thyroid deficiency in children.

This clear demonstration of pathological changes in the tissues of a baby three months of age gives a rational explanation for the established fact that hypertension or atherosclerosis may begin in childhood. Both diseases occur frequently in some families and not in others. The same is true for hypothyroidism. It would appear that the low thyroid function even in an infant can start the pathological damage to the blood vessels by depositing the mucopolysaccharides in the walls of the arteries. This process could be the forerunner of the accelerated atherosclerosis which has been demonstrated in all of the arteries of thyroidectomized animals and in humans with low thyroid function.

Hypertension (high blood pressure) is closely related to kidney function. Goldblatt[30] produced fatal hypertension in dogs many years ago by placing on the kidney artery a simple metal clamp that could

be tightened periodically to restrict the flow of blood through the kidney. In hypothyroidism there is a reduction in blood-flow through all the organs including the kidneys. Atherosclerosis in the kidney artery could further reduce the blood-flow as effectively as Goldblatt's clamp. Hypertension is very common in hypothyroidism. Barnes[31] has recently reported that thyroid therapy alone was found to relieve 80 percent of the elevated blood pressures seen in a general practice during the past 25 years. Children from patients with hypertension have been found low in thyroid function, and thyroid therapy in the offspring with hypertension has been quite efficacious.

The observations of Andersen leave little doubt that thyroid deficiency can be a potent factor in the genesis of atherosclerosis and hypertension in children. Obviously, if prevention is to be achieved, one must look continually for hypothyroid symptoms from the time of birth. The earlier a diagnosis of hypothyroidism can be made and appropriate therapy started, the better will be the chances of avoiding a premature death from a heart attack.

CHAPTER IV

Symptoms of Thyroid Deficiency

My interest in the thyroid gland grew out of necessity rather than choice. The late Professor of Physiology, Anton J. Carlson, arbitrarily assigned this subject for my doctoral thesis when I entered his department at the University of Chicago over 45 years ago. I would have preferred another subject, but during the depression one was so happy to find any job permitting the continuation of education that no objections were raised. As time passed, the wisdom of this great educator's decision was more than justified. He was aware that many secrets of this tiny gland remained undiscovered, but I am sure that he had no idea that this humble beginning would solve so many of the problems in medicine. Heart attacks had not appeared on the horizon at that time. Neither of us foresaw the solution of this major problem of the century.

My duties included teaching the medical students a class in endocrinology on the mysterious glands of internal secretion. One of the glands would be removed from an experimental animal, and the students would follow the development of disease caused by the glandular deficiency. If the pituitary were removed, growth and development ceased, and many organs of the subject malfunctioned. Removal of the parathyroid glands led to muscular contractions as a result of the drop of calcium in the blood. If the pancreas were removed from a dog, diabetes developed at once, and the students could follow the treatment. Removal of the adrenals soon led to disturbances in the mineral metabolism and the blood sugar; death promptly occurred. The role of the ovary in the menstrual cycle and reproduction was studied in the white rat where the cycle is repeated every four days.

However, the most dramatic story was the loss of the thyroid in baby rabbits only 3 weeks of age. Within 2 weeks, growth was retarded, the fur became dry and brittle, the ears drooped, and muscles became weak with the development of a pot-belly. Bone development was

abnormal, and body temperatures were subnormal; they did not develop sex characteristics, were sterile, and they seemed to lack normal intelligence. They became sluggish and did not play together as normal animals do, they became anemic, were susceptible to repeated respiratory infections, and died at an early age with pneumonia. Animals or humans presenting these bizarre symptoms are called cretins. It was apparent that every cell in their bodies was affected by loss of their thyroid glands. These results are not surprising, for although it is still not known all that the thyroid hormone does, it certainly controls the rate at which each cell burns the food that gives us energy. Hence, every part of the organism is affected by loss of this important hormone.

After teaching this course and doing extensive research on the loss of the thyroid hormone in experimental animals for five years, I then finished medical training and began its practice. Past experience of having seen such bizarre symptoms disappear with thyroid therapy in animals alerted me to look carefully for similar ailments in the human. It soon became apparent that many patients were going from doctor to doctor with a variety of seemingly unrelated complaints; accordingly, with no definite diagnoses made, they were called "hypochondriacs."

Soon after entering private practice, I encountered such a patient. She had been through a well-known midwest clinic with multiple complaints but with no definite diagnosis. Her blood pressure was low, and fatigue was an outstanding symptom. She was told that she was suffering from neuro-circulatory asthenia and that she should go home and get used to it. Many of the symptoms seen in the baby rabbits were present including a low Basal Metabolism. Thyroid therapy gave her a new lease on life with elimination of most of her complaints. Neuro-circulatory asthenia is seldom seen in the literature today; in its place has arisen a new, wider connotation, "psychosomatic disease." If the physician does not recognize the illness at once, he assumes that the complaints are figments of the patient's imagination. The patient is hurried off to the psychiatrist to re-orient his thinking. The best that the psychiatrist can do is to teach the patient to tolerate his symptoms and live with them. Occasionally a patient is told that his symptoms are not mental.

One of America's greatest physicians, Sir William Osler, contended that if the physician would let the patient talk long enough, the patient would make the diagnosis. This is as true today as it was at the turn of

the century. Having seen that bizarre symptoms could be relieved in the cretin rabbit by thyroid therapy, I was prepared to try thyroid therapy for similar symptoms in the human. During 40 years of clinical observations, I have found relatively few patients suffering from psychosomatic disease. It is reasonable to suspect that a clue has been missed in those not showing organic changes. Some of my contemporaries feel that 90 percent of their patients are plagued with mental disorders. There has been a steady stream of these coming to my office; in most cases thyroid deficiency has been overlooked. Some colleagues are convinced that it is I who has psychosomatic disease, but if relieving patients' symptoms with thyroid therapy will cure their miseries, I shall be glad to carry the accusation to my final resting place.

Observations on the baby rabbit left no doubt that symptoms of thyroid deficiency might originate any place in the animal. Years of experience with the human has reasserted that the same is true for man. Symptoms may appear soon after birth, or they may be recognized first in old age. No age is exempt, nor is any part of the body: a headache on one end may be just as significant as cold feet at the other extreme. Skin infections on the exterior may signify a lack of thyroid, while just as important is the presence of anemia, indicating that the bone marrow at the center of the bones is malfunctioning. Growth may have been retarded, and the individual may have a short stature; yet, in another patient growth may have continued too long, and a seven-footer resulted. As strange as it may sound, both are due to a lack of thyroid. Paradoxes are common in hypothyroidism; for each, there is a rational explanation.

Since the newborn cannot talk, symptoms must be picked up in other ways. Here is another paradox: the baby may sleep all of the time and may require being awakened for feeding, or on the contrary, sleep may be frequently interrupted with crying and irritability. The mother may be very happy that her baby sleeps all night as soon as she arrives home from the hospital, but this is a danger signal; she should check the Basal Temperature. A markedly thyroid-deficient baby must be treated before 6 months of age since this is the period of rapid development of the brain, and without adequate thyroid, permanent mental retardation may occur. The tongue of the baby may appear too large for the mouth and may protrude through the gums. This alone will make the diagnosis when present, but like all symptoms, it is not always in evidence. The child who is slow in cutting teeth, slow in talking and

even slow in learning to walk is always suspected of being low in thyroid function. The muscles and tendons do not develop proper strength; if this is not corrected, even in adult life, there is abnormal mobility of the joints. In the circus, the freak who can tie himself into knots owes his talents to a thyroid deficiency. Skin diseases in babies and children are usually eliminated by thyroid therapy whether they are due to infections, dry scaly skins, or eczema.

A susceptibility to respiratory infections may be present at birth and continue to old age. It is often more important to the preschool child, although exposure to other children may be increased in the classroom. Not only are the common colds frequent, but the complications, such as tonsillitis, sore throats, middle ear infections, sinusitis, and pneumonia depend to a great extent upon the resistance of the patient. The hypothyroid individual can raise the resistance remarkably by thyroid therapy in about two months, but the benefit is lost after six months if therapy is stopped. Removal of the tonsils will stop tonsillitis, but does nothing about the resistance to the other complications. Certainly more than half of the initial infections can be prevented with thyroid treatment, and the complications are rarely encountered after the resistance is built up.

In severe thyroid deficiency, the resistance may be so low that the antibiotics are relatively ineffective. I have never seen a death from a middle ear infection, yet at Graz, Austria, where the entire population has been low in thyroid secretion for centuries, deaths from middle ear infections are still prevalent in spite of antibiotics.

Pneumonia seldom occurs in patients on adequate thyroid therapy. Furthermore, a patient with a history of pneumonia in the past will usually have other symptoms confirming the presence of low thyroid function. Resistance against the virus diseases is also increased by thyroid administration. Among the senior citizens influenza shots are not necessary if the thyroid function is normal.

The susceptibility to infections is not limited to the respiratory tract. The urinary tract is a favorite site for repeated infections, and each attack will cause some damage to the kidney whose function is gradually lost. During the past 30 years routine thyroid therapy to this group of patients has completely stopped the loss of kidney function with uremia and the requirement for kidney transplants. The vaginal tract is another favorite area for infection, and the treatments may be unsatisfactory unless the resistance of the tissues is raised by thyroid.

Pinkeye or infections around the eyeball are more common in the patient with low thyroid activity, and styes are likely to occur in the hair follicles of the eye lashes.

The cold hands and feet of the hypothyroid patients signify poor circulation to the skin which results in a susceptibility to skin infections. The amount of blood going through the skin per minute may be only 60 percent of the normal quantity. This accounts for the chilly feeling and a desire to be in a room uncomfortable to normal people. A variety of skin infections such as impetigo, erysipelas, boils, and several others are common. Prevalent pimples of adolescence are due to a disturbance in fat metabolism in the skin with superimposed localized infection. Over 90 percent of the acne patients clear up on thyroid therapy since the circulation is increased and the fats are better metabolized. Older patients in whom acne has persisted are usually low in thyroid function. A male of 61 came in after having had a heart attack. His back was covered with acne which disappeared as soon as he could tolerate small doses of thyroid to discourage the atherosclerosis. There are very few patients with skin diseases of any kind who will not be benefited by thyroid. More than one-half of the cases of psoriasis respond favorably. One of the most serious skin disorders is lupus erythematosis. Many years ago the British discovered that the form affecting only the skin clears nicely with thyroid. Another variety may affect the internal organs causing changes similar to arthritis. I have found that a combination of thyroid and a small dosage of prednisone are very useful in these patients.

The reproductive organs need a correct supply of thyroid hormone to function properly. Long before thyroid hormone was available for treatment, it was known that in the goiter regions the reproduction diminished, and entire families disappeared. Any abnormality in the menstrual cycle is an indication for a Basal Temperature check. Some girls start their periods early — one of my patients at the age of five. On the other extreme are those who begin beyond the age of fifteen. Both groups are low in thyroid, and proper dosage will usually correct the condition. Irregular periods are common; some girls may have two periods a month while others may skip several months between periods. Profuse flowing is not unusual in the thyroid-deficient girl; clots of blood may form in the uterus and their expulsion causes cramps as severe as childbirth. More than 90 percent of the menstrual disorders are improved with adequate thyroid therapy.

The most frequent cause of sterility is a lack of thyroid hormone. The Basal Temperature should be checked on both wife and husband before the more sophisticated tests and expense are undertaken. One of my happiest couples had tried in vain for 17 years to have a family. As soon as the husband was seen, it was realized that he also needed thyroid therapy. She had her first baby at age 39, and two years later carried another uneventful pregnancy to term.

Miscarriages are more often due to a lack of thyroid than to any other cause. One of my patients had had 15 pregnancies and only five babies. Her history left no doubt that she had been an unsuspected hypothyroid most of her life. When she appeared, it was a little late for any help with reproduction; she was 65 at the time. However, a high percentage of women with histories of repeated miscarriages can usually carry babies to term under appropriate thyroid therapy, but during the pregnancy the dosage may have to be increased because of the extra load on the mother.

Toxemias of pregnancies, including high blood pressure, protein in the urine, and marked retention of water are usually due to a lack of thyroid. During 20 years of obstetrics, by watching carefully for evidence of hypothyroidism, only one case of toxemia was seen. In that instance, her first visit was a week before she was due to deliver. The blood pressure was high, she was swollen with fluid, and the urine was loaded with protein. Usual treatment was instituted, but she went into convulsions three days later. An emergency Cesarean-section fortunately saved the lives of both her and her baby. This is one place where an ounce of prevention is worth far more than a pound of cure.

There would be much less gynecological surgery if the thyroid function of the female were balanced during her lifetime. The average physician may have 20 candidates annually for dilation and scraping out of the uterus to stop abnormal menstruation. In 20 years not one has been necessary in my practice. Within a period of six months I have seen three women below the age of 25 in whom the uteri had been removed to stop vaginal hemorrhage. In each case the history and physical examination indicated that probably thyroid therapy would have controlled the menstruation and allowed each to raise a family rather than to remain childless. Ovarian cysts are common in the thyroid-deficient patient; if detected early, they will usually disappear on thyroid administration. Fibroid tumors of the uterus are rare in

women with normal thyroid function; hence, prophylaxis is the course to follow here.

Babies who are born dead at term or those who die shortly after birth have been a mystery. Also of great concern is the so-called "crib-death" in which the baby appears normal for one to two months then is found dead in the crib with no apparent illness. I have seen only one such case, and in that instance the mother was low in thyroid function. In domestic animals, such as the swine, a high death rate among the newborn occurs if the diet of the sows is low in iodine. A mother giving birth to a dead baby or to one who succumbs to a crib-death should take her Basal Temperature and be put on thyroid if indicated. The viability of both a mother and baby will be increased by thyroid therapy if her thyroid function is subnormal.

Some patients may have only one or two symptoms of a thyroid deficiency while other patients may have several complaints. The majority, however, will have some degree of fatigue. This may be manifested by a requirement for more sleep: after as much as 9 or 10 hours they may feel as tired in the morning as they were before retiring. Some patients will fall asleep if they sit down for a few minutes, even to watch TV. Routine duties that are easily accomplished by the normal individual may overwhelm the hypothyroid. This is called the "tired-housewife's syndrome" and is usually considered psychosomatic in origin. Still, the same patient on adequate thyroid therapy — without the fatigue — can efficiently take care of her duties and have enough energy to engage in outside activities. Her depression is soon lost, and she enjoys what she is doing.

An entire book could be written on the mental problems associated with thyroid deficiency. These may begin with the temper tantrums of the infant, the fears of the small child, the failure to adapt to the routine of the family or the schoolroom, or anti-social behavior of the teenager, incompatibility of a marriage, failure to hold a job, or even in old age the ideas of persecution or the lack of memory; often these are relieved by thyroid therapy. The child who does poorly in his school work is unable to concentrate; the attention-span is short, and although his intelligence may be above average, he fails and becomes depressed. For three years I saw all of the failing students at an engineering school. The curriculum was difficult. Those students in attendance represented "the cream of the crop" for only those in the

upper 10 percent of the high school classes were accepted. Nevertheless, a few would fail, sometimes in the first year or even in the final year. They would become depressed; one attempted suicide. Fortunately he was not successful, and when his thyroid was adjusted, he graduated at the head of his class. No failing student in this group missed showing symptoms and physical findings consistent with thyroid deficiency, and each responded satisfactorily when placed on therapy. My personal feelings are that if thyroid deficiencies are recognized in children and treatment is continued throughout life, most "psychosomatic complaints" will disappear.

It is obvious that symptoms may originate anywhere in the body; in fact, thyroid deficiency may mimic any disease. Only two more conditions will be mentioned, but remember that it is well to take one's Basal Temperature regardless of what the symptoms may be.

The low temperature is the most common cause of anemia. The red cells are formed in the bone marrow in the spine and in the upper parts of the arms and legs. The temperature of the extremities progressively decreases towards the hands and feet; no blood is formed in the cool part of the bones. This is easily demonstrated by using the white rat's tail. Curving the tail around and suturing the tip inside the abdominal wall will lead to blood formation in the tip at the higher temperature, while the loop of the tail will form no blood. The patient with susceptibility to anemia should check the Basal Temperature. None of the treated patients need extra iron.

Migraine headaches are disabling at frequent intervals. They run in families just as hypothyroidism does. The fatigue may alter the fluids in the body, and the tissues swell. These patients learn that at the time of a headache their shoe-laces must be loosened because the feet expand. The brain cannot expand, and the pain may even lead to vomiting. The patient goes to bed, the rest eliminates the swelling, and recovery occurs. Thyroid therapy will raise the threshold of fatigue, and about 95 percent of the migraines can be avoided. Excessive fatigue may cause a recurrence at any time. Headaches from sinus infections are far more common, and some improvement in these will occur if the resistance to infection is raised with thyroid.

Regardless of your complaints, take your Basal Temperature; maybe "you are not so hot."

CHAPTER V

Effective Prevention of Heart Attacks Over a Forty Year Period

For 13 years, thyroid-deficiency problems occupied my time. Patients with such complaints were numerous and their gratitude for finding some relief was rewarding to me. Suddenly in 1950 a new tragedy entered my practice. I saw my first patient with a heart attack. I knew very little about this new "epidemic" that was sweeping the country. Although my medical education had been at Rush Medical College in Chicago where Professor James B. Herrick in 1912 had described the successful treatment of cases of sudden collapse, this disease was so rare when I was in school that very little time was devoted to it. We spent many hours on rheumatic fever, congestive heart failure, hypertensive heart disease and other cardiac problems, but my notes are almost devoid of coronary disease. In the meantime, I had been too busy to keep up with the literature on a disease which I had not seen. Now a new nightmare was to haunt me.

This heart attack had occurred in an acquaintance who had not been a patient on thyroid therapy. The electrocardiograms were submitted to a cardiologist in a distant city who reported that the artery on the back of the heart was occluded on the first day, followed by a second attack the next day with occlusion of the artery on the front of the heart. The consultant gave little hope of recovery. However, the patient surprised everyone, and was able to return to work in a job requiring considerable responsibility. After it had become evident that thyroid therapy should be beneficial, he was started on treatment. He did very well for 9 years, but died from a second attack at the age of 68 after having omitted his thyroid medication for several months. I had moved to another state, and he had become negligent.

Obviously my interests had to be widened to include another major disease. Why had I seen no heart attacks during the previous 13 years during which interval this disease had become number one in importance? Had I been preventing heart attacks with thyroid therapy

so commonly employed in my practice? It seemed incredible that this could be true. Yet the thyroid had been involved in so many diseases in which it was unsuspected that the possibility could not be lightly discarded.

Elevated cholesterols had come into the limelight and were being accused as the culprit in heart attacks. I well remembered that the thyroidectomized rabbits had elevated serum cholesterols. The work of several investigators had demonstrated that the elevated cholesterols could be brought down to normal with thyroid therapy. I had a backlog of patients who had been on thyroid for 13 years. New patients with the symptoms of thyroid deficiency were arriving every day. How was thyroid administration affecting their cholesterols? No one was in a better position to investigate this problem; hence, a new field of endeavor was initiated. It was fully realized that many years of patience and hard work would be necessary to determine the role of thyroid deficiency in heart attacks.

Subsequently, the routine examination included an x-ray for heart size, an electrocardiogram, and a battery of blood tests. Periodically at one- to two-year intervals the tests were repeated so that any changes would be evident. In 1951 the controlled study by Dr. Kountz using thyroid therapy, as mentioned in Chapter 3, added interest and effort to the project. It seemed reasonable that if vascular deaths could be delayed in elderly people, earlier treatment should be even more effective.

Some valuable lessons had been learned from the five-year association with the late Professor Carlson. One of his requirements for a legitimate experiment was to alter only one factor at a time. Accordingly, if we were to study the influence of adding thyroid, all other conditions should be left as closely as possible to their previous state. Hence, no one was taken off cigarettes. Alterations in the diet were discouraged; this was often difficult due to the propaganda to the laymen. Extra exercise was discouraged and when jogging became fashionable, patients were told that time would prove the folly of it. Disease is not cured by exercise; when a patient becomes ill he is put to bed to conserve energy for fighting the disease. Tuberculosis was conquered by bed-rest and not by jogging. If one is going to be engaged in fighting or must run to keep out of fights, there is an excuse for being in first class condition. Otherwise, *exercise for pleasure is commendable,* but no one has proven that it will prevent disease.

A review of the literature mentioned in Chapters 1 and 3 left little doubt that thyroid therapy would decrease the incidence of heart attacks. So strong was the suggestion that if half of the patients had been put on therapy and the other half kept as controls, the physician's conscience would have been disturbed for not bringing the most promising therapy to the benefit of each patient. All of the other methods of approach were being employed by many institutions and practitioners. It was certain that comparisons could be made in time. The Public Health Service started the Framingham Study in Massachusetts where more than 5,000 residents were studied by techniques very similar to those being used in the present investigation. The essential difference was that patients in Framingham were probably receiving alterations in diet and other modes of living because of their proximity to Harvard. Publications by the Framingham Study[32] in 1957 confirmed observations of previous workers that hypertension, high cholesterol levels, sex and advancing age were important factors increasing the incidence of heart attacks. In younger individuals, males were affected much more often than females, but in the older-age groups, as more men died off, the females passed the "stronger" sex in deaths from heart attacks.

The Framingham Study included over 5000 patients between the ages of 30 and 62. They represented the majority of residents in a typical small American town. At the time of the initial examination, there were 76 cases of atherosclerotic heart disease. The purpose of the study was to uncover risk factors in heart attacks. After 20 years of the scientists' time and millions of dollars of the tax payers' money, no new risk factors were uncovered. The normal occurrence rate of new heart disease was established. At a relatively constant rate which did not improve in spite of all the propaganda about smoking, diet, exercise and unsaturated fats, over 30 new cases of heart disease were uncovered each year. This amounted to more than 600 in the 20 years, or an average of 150 per 1000 patients.

In my thyroid-treated group using the same methods of examination and similar criteria for detection, only 4 new cases were encountered for the total 20 years on over 2000 patients or an annual rate of 2 per 1000 patients. It would appear that 75 times as many new cases of heart disease developed in Framingham in spite of the fact that many of them had adhered to the advice about diet, smoking, exercise, etc.

One can never be sure how many patients follow the physician's advice. The marked increase in the sale of unsaturated fats and the ruination of the dairy industry leave no doubt that many patients did alter their diets. We have no way of knowing how many in the Framingham Study altered their diets, nor do we know how many of my patients ate polyunsaturated fats. Some of them did continue with the animal fats; 62 percent of the male population on thyroid smoked cigarettes. The fact remains that the big difference between the two groups was that my patients were on thyroid therapy.

At the end of 20 years the results of thyroid therapy were compared with those of the Framingham Study. At that time, 1569 patients had been treated with thyroid and could be compared with those in Framingham. Females below the age of 60 years, without any elevation of the blood pressure or cholesterol in the serum, had the least danger of developing new heart disease. In this category 490 patients had received thyroid therapy in the present study. Over the period of observation, one might have expected more than 7 new cases of heart damage, but none were observed. In the present study, there were 172 women below the age of 60 with hypertension and/or high cholesterol readings. These cases are more susceptible to atherosclerosis, and they should have added more than 7 additional new cases of heart disease. None appeared in the thyroid-treated patients. Women between the ages of 60–62 are far more susceptible to heart damage, and although one would have expected to see 8 new cases in the 182 patients in this category, none occurred.

Men are more susceptible than women, and continuing the comparison with the Framingham Study, in men below the age of 60 without any high-risk factors, one would have expected 13 cases of new heart disease in the 382 subjects; only one case occurred. There were 186 men below 60 years of age with elevated serum cholesterols and/or hypertension. This group should have produced 19 new cases of heart disease. Only two were detected. For the men between 60–62 years of age, the risk is the highest of all. Among the 157 men in this group, one would have suspected 18 patients to develop new heart disease. Only one case occurred.

It is apparent that the thyroid therapy has been remarkably effective in preventing new heart disease in all age groups and in both low- and high-risk subjects. Undoubtedly, the patients in Framingham were encouraged to correct their style of living, alter their diets, reduce

their weights, stop smoking and get more exercise. None of these were recommended in the thyroid-treated group. There seems little doubt that the thyroid therapy reduced the rate of formation of atherosclerosis in the arteries of the hearts.

Previous chapters have indicated that thyroid deficiency is a potent factor in the origin of heart disease. Personal experience indicates that about half of the population suffers from some degree of thyroid deficiency. All of the patients in the present study were low in thyroid function and benefitted from therapy in many other ways in addition to less heart damage. One might have expected twice as many new cases of heart disease in the present study as were found in the Framingham Study, if the thyroid therapy had not been efficacious. Yet actual comparison of the 4 cases observed with the 72 expected from the Framingham Study shows that 94 percent of new patients with heart disease were avoided. No other suggestions for preventing heart attacks have approached these results. They deserve the attention of those interested in reducing the carnage among the younger population.

Another five years have passed since the above studies were published.[5] The number of patients on thyroid therapy for the prophylaxis of heart attacks is growing rapidly, and now more than 2000 are included. The success has not diminished. Seldom does a new case of heart disease appear. It is only during excessive stress that symptoms and laboratory confirmation of a new heart attack occurs. Stress will be discussed in a later chapter.

These results are very similar to those of Dr. Kountz. The essential difference is that he ran controls simultaneously, and followed only the deaths in the two groups. In his study thyroid therapy was more efficacious if it were started in younger patients. That was true in the present study since the youngest patient developing new heart disease was 56 years of age. His father had died with a heart attack at age 54. Hence, it is reasonable to assume that atherosclerosis had been progressing in the patient for many years before he was started on thyroid. He was first treated at the age of 51, and in retrospect, the dosage may have been inadequate. He was only on two grains daily, and had not been rechecked in the meantime. Kountz pointed out that even in older patients up to 67 years of age, thyroid therapy would reduce the incidence of vascular accidents. The rarity of new cases in the present report is probably best explained by the fact that many of the patients were younger when therapy was started. From Andersen's

report, in the last chapter, it is obvious that the atherosclerosis starts in the hypothyroid infant, and early therapy should be more efficacious.

Another clear indication that thyroid therapy has a place in preventing heart attacks comes from the dropouts in the present study. It is difficult for young people to believe that they are susceptible to serious disease. Their pride is hurt in having to take pills to prevent something that they have every hope of avoiding. Consequently, more than 30 individuals discontinued therapy against advice that they continue. Fatal heart attacks occurred in each within a six-year period. Thirteen of them were under 60 years of age at death, with two in their 20's, one in his 30's, two in their 40's, and eight in their 50's. These corresponded to the early deaths seen in the Framingham Study, and were not found in those who continued their thyroid therapy. There seems little doubt that the young hypothyroid patient is unusually susceptible to heart attacks. This is confirmed by the fact that 13 of the 30 fatalities were under 60 years of age. This premature mortality is considerably higher than that reported in Framingham. All of these dropout patients in the present study had been diagnosed and treated for hypothyroidism. The evidence indicates that an effective prophylactic program of small doses of natural thyroid can markedly reduce the carnage from heart attacks in young patients.

The clearest indication of the efficacy of thyroid comes from the treatment of the patient who has had a previous attack. In this instance, there is no question about the susceptibility of the individual, since a premature heart attack has occurred. This was the group of patients upon which the British tried polyunsaturated fats and found that diet had no effect on recurrences of heart disease. It was also this type of patient that the Coronary Control Group wasted more than five years and much expense with female hormones, choloxin, clofibrate and niacin on over 8000 patients. None of these compounds reduced the incidence of recurrent heart attacks, and there had been no experimental work to indicate that they should. All of the chemicals reduced the blood cholesterol some, but as so clearly pointed out in the final chapter, cholesterol has nothing to do with heart attacks. The cholesterol is elevated in many patients suffering heart attacks, but the thyroid deficiency is responsible for the deposition of mucopolysaccharides in the arteries. These mucopolysaccharides and not the cholesterol cause atherosclerosis.

How unfortunate that the designers of the Coronary Drug Project did not include desiccated thyroid among the agents lowering cholesterol levels in the blood. Animal experiments and all of the other evidence on the human, presented in the last chapter, have clearly indicated that this would be the physiological approach. Solving the heart attack problem by any means other than diet or reduction in cholesterol would put the designers in an uncompromising position. Their careers have been built on the cholesterol theory, and they are unwilling to admit that they have been in a blind alley. In February, 1976, they announced that a new super experiment would be tried (*Family Physician,* Vol. 13, page 132) on 12,000 patients, half of whom will be encouraged to stop smoking, to reduce the fat in their diets and to follow treatment for their hypertension. The article states that other large-scale studies have begun using aspirin and aspirin plus another synthetic compound on patients who have recovered from heart attacks. How long must this witch-hunt go on before the taxpayers' money can be directed toward something more promising?

The Coronary Drug Project and the new Multiple Risk Factor Intervention Trial cannot plead innocence about the benefits of thyroid therapy. In January, 1962, one of the authors of the above projects sat next to me at the meeting of The Oregon Academy of General Practice in Portland, Oregon. The symposium was on "The Causes and Treatment of Atherosclerosis." I presented thyroid therapy as outlined in this book. He talked about estrogen treatment. Less than two hours after he delivered his presentation, he heard my lecture. In the intervening years he has never mentioned my work, but he has buried the estrogen therapy.

The only excuse for not using thyroid therapy in some of the multiple mass studies may be another mistake in dosage comparable to the 1938 scare. For a time, Gofman and his associates[33] felt that the elevated beta-lipoproteins were responsible for the damage to the arteries in atherosclerosis. They suggested the use of thyroid for lowering them. To test the prolonged effects of thyroid therapy on the beta-lipoproteins, they administered to mental patients sufficient thyroid to completely suppress their own secretion. It took 5 grains daily, and the authors concluded that thyroid therapy could be used. Some clinicians interpreted their findings to mean that 5 grains daily could be used in patients with heart damage. This only confirmed the observations of many physicians, reported prior to 1938. Had they used one

or two grains daily, as had been suggested, their results would have been more than encouraging and their disappointments would have been avoided.

Over the past 25 years, rarely has a patient on thyroid therapy had an initial heart attack. Many patients have been seen with a history of heart attack and hospitalization prior to seeing me. They have been put on small doses of thyroid, usually an initial dosage of one-half grain daily. After one or two months, the dosage has been increased one-half grain at a time, and in some cases, over a period of six months, has attained as much as 2 grains daily. What results have been obtained by this cautious approach to the use of thyroid after a heart attack? The number treated with thyroid in the present study is too small for statistical analysis, but during the past 25 years a death among these patients has rarely occurred. Most of them are surviving the normal life span, and when a second attack does occur, it usually can be attributed to some exceptional stress. This rare occurrence among those on thyroid is in sharp contrast to a mortality of 5 to 10 percent annually among patients with a previous heart attack treated by conventional therapy.

The fact that stress may overcome the beneficial effects of thyroid is illustrated by the following example. A 44-year-old male had his first attack in July of 1962 followed by three more, each proven by hospitalization, before I saw him in May 1963. Thyroid therapy was started cautiously, and the anticoagulants (blood thinners) were stopped. His dosage was gradually increased to 2 grains daily. He did very well and was back at work. He was convicted of a felony, and a penitentiary sentence was imposed. The stress of the trial and his insistence of innocence was overwhelming, and he died from a fatal heart attack in July of 1967 before incarceration. From his improvement during the previous four years, he should have survived much longer without this unusual stress.

Personal observations have fully confirmed and extended the excellent controlled studies of Dr. Kountz. Apparently thyroid therapy has no influence on the advanced arterial damage. A few autopsies on patients who had occluded coronary vessels prior to starting the thyroid revealed that the vessels were still closed; but the relief of hypothyroidism seems to stop the deposition of the mucopolysaccharides and the further progression of the atherosclerosis. If we can learn to diagnose the susceptible ones in childhood and keep the thyroid hor-

mone level near the normal requirement during the individual's lifetime, it would appear that a heart attack can be delayed until advanced age when fair wear and tear may end one's misery. The most susceptible patients are the major problems at the present. Since they represent only 27 percent of the total heart attacks, it seems reasonable that thyroid therapy may be able to eliminate these untimely deaths. There is adequate evidence that more than 27 percent of the population are deficient in thyroid hormone. Further evidence indicates that these individuals are depositing mucopolysaccharides until thyroid therapy begins.

Other investigators have also confirmed the work of Kountz. Dr. Murray Israel,[34] in New York, began treating cases of advanced atherosclerosis with thyroid therapy before I entered the field. He found, as others had previously, that the requirement for vitamin B is increased when the metabolism is raised. During periods of growth, pregnancy and any other condition with an elevation in thyroid secretion, more vitamin B must be administered or symptoms of a deficiency appear.

With the usual therapeutic doses of desiccated thyroid, Israel has added an excess of several vitamins. To hasten the utilization of fat and lower the cholesterol, he has added another tablet containing Choline, Inositol and Pyridoxine. As many as 30 of these tablets daily may be administered. In addition to the oral therapy, frequent intravenous injections of still another preparation composed of synthetic thyroxine, synthetic vitamin B-12, calcium gluconate and gelatin are given.

As far as can be judged from the published reports, a single appropriate dose of oral thyroid supplemented with Brewer's Yeast tablets, for those in whom more vitamin B is needed, has given about the same results as the more expensive method of Dr. Israel. Since Friedland's demonstration in 1927, repeatedly thyroid therapy has corrected the elevated serum fats and cholesterol in most cases. In hundreds of patients treated in the last 25 years, 95 percent of the cholesterol levels have returned to normal with only thyroid therapy. No premature heart attacks have occurred in the 5 percent whose elevated cholesterols persisted in spite of thyroid therapy. As pointed out frequently in the present report, atherosclerosis results from the thyroid deficiency and not from the elevated serum fats that accompany it.

Wren[35] in two reports has confirmed the efficacy of thyroid therapy in cases with coronary heart disease. A total of 347 patients with evi-

dence of heart disease were studied. Only 9 percent of them were low in thyroid function according to standard tests, but all were treated with thyroid since it was realized that the thyroid function tests did not reflect the clinical condition. The serum cholesterols fell an average of 22 percent. In 41 patients, angina was present at the start of the experiment. No new cases of pain appeared during thyroid administration, in 29 less pain and more exercise tolerance occurred, while in 12 there was no change in angina. The expected mortality over a 5-year period was reduced by 58 percent. His results are quite similar to those of Kountz in a similar-aged population. In a small series of patients, Wren also injected synthetic thyroxine in a manner similar to that used by Israel, but could see no difference in the results from those receiving only oral thyroid therapy.

The consistent results of numerous authors over the past 40 years leave no doubt that thyroid therapy is efficacious in delaying fatal heart attacks. Since the process begins in childhood, the earlier the diagnosis, the greater will be the benefit. Often patients with low thyroid function will have symptoms originating anywhere in the body. If each of these symptoms is treated at its first appearance, health of the individual will be greatly improved. A fringe benefit will be a reduced danger of a premature heart attack.

CHAPTER VI

The Fallacy of Thyroid Function Tests

When I began writing prescriptions 40 years ago, it was difficult to accurately diagnose thyroid deficiency. Time has not changed the problem. For years only the Basal Metabolism test was used. "Basal" means that the patient has been resting for several hours, no food has been consumed, no exercise has been taken, and no excitement nor tension are present. Each of these factors raises the oxygen consumption. An attempt is made to measure the minimum amount of oxygen necessary just to sustain life's processes. Hence, the ideal time to make this measurement is immediately upon awakening after a good night's sleep.

The apparatus necessary for the Basal Metabolism consists of a tank of oxygen, a motor to circulate the oxygen to the patient, and tight-fitting rubber connections to prevent the escape of oxygen. A clothespin-like clamp is placed on the patient's nose, a rubber tube is stuffed into his mouth, and he is told to "just relax." Thereby arises a problem. A patient with claustrophobia tears the mask from his face, convinced that he is about to suffocate. A patient under tension may double his oxygen intake. Obviously, this apparatus is not available in each home, nor is a trained technician waiting in the wing for the patient to wake up. If the patient spends the night in the hospital, tension is present. If the patient sleeps at home and upon awakening, goes to the hospital or laboratory, this activity raises the oxygen consumption. In spite of all these difficulties, the Basal Metabolism gives results as accurate as the modern functional tests.

In the early days the physician obtained a careful history, made a detailed physical examination looking for confirmatory evidence, and did not let the Basal Metabolism overpower his judgment, if he thought the patient needed thyroid therapy. Often he used the therapeutic test. He knew that small doses of thyroid would do no harm; a short trial frequently confirmed his suspicion. The patient was very happy to see his symptoms disappear.

Scientific medicine came to replace the art. Laboratory tests were devised to aid the doctor in his diagnosis. This is an excellent idea, if a specific test can be elaborated, but unfortunately the "tail began to wag the dog." At the present time, out of 30 tests being used, not one gives an accurate index of the amount of thyroid hormone necessary for health of the patient. The blood tests measure the amount of hormone circulating in the blood stream, but the thyroid does not exert its influence in the blood. The blood level only guarantees the presence of an adequate supply in case a cell, far distant from the thyroid gland, suddenly needs more thyroid hormone. The thyroid hormone performs its duty on the inside of each of the billions of cells all over the body. We have no means of measuring the concentration of hormone in each of the cells.

Regrettably, medical students are being taught that, unless the blood level of the hormone is low, the patient does not need thyroid therapy. The use of symptoms, the corroborating physical findings, and common sense have been discarded. *The end-result is that thyroid deficiency is the most common illness entering the physician's office, and it is the diagnosis most often missed.* This opinion was expressed by Dr. A. S. Jackson,[35] a thyroid specialist in Madison, Wisconsin, in the *Journal of the American Medical Association* in 1957; it is just as true today as it was then.

The multitude of thyroid tests do not justify space for a detailed discussion here, but enough will be presented to show the reader that he cannot rely on thyroid-function tests. If he has symptoms for which the physician can find no diagnosis, he will do well to continue searching for a doctor who realizes the fallacy of thyroid-function tests.

The first chemical blood test measured the Protein-Bound-Iodine, called the PBI. It came into use about the time that I began to practice. The theory for it was excellent: iodine was present in the thyroid hormone which was bound to protein in the blood. I soon learned that some patients with clear signs of thyroid deficiency had normal PBI readings. Furthermore, treating these same patients with thyroid cured their complaints; the PBI was still in the normal range. This happened so frequently that I never adopted the PBI test. Most of the profession went overboard for it; some of the most able thyroid specialists "swore by it," but I continued to "swear at it." It took 35 years to win the argument, but eventually all investigators agreed that the PBI could not be used to diagnose thyroid disease.

It is shocking to think of the thousands upon thousands of patients during this interval who were denied therapy because of a faulty laboratory test. It was during this same 35-year-period that psychosomatic complaints became so prevalent. Having seen hundreds of these patients, who had symptoms of thyroid deficiency and whose complaints were relieved by thyroid therapy, I have little doubt that these patients should have been referred to someone understanding thyroid problems rather than to a psychiatrist "for psychosomatic complaints." Some psychiatrists have become aware of the frequency of thyroid deficiency among their referrals, and have found that thyroid therapy has solved many problems.

A rash of new thyroid-function tests began to appear. Obviously none of them were reliable or there would be no necessity for finding a better one. I never order the thyroid-function tests since two or three are commonly employed, and they may disagree with one another. I am not smart enough to know which one to believe, so why waste the patient's money?

An experience is worth recording. One of the commercial firms wanted to market a new product of thyroid hormone. They asked me to compare the effect of substituting the new product in place of natural thyroid which had satisfactorily controlled symptoms in 40 consecutive patients. They requested that each of the 40 volunteers should have three thyroid-function tests *before* the medication was changed. The initial samples illustrated the unreliability of the thyroid-function tests. All of the patients were free of symptoms and had been for prolonged periods. In 20 percent of the cases, *a single specimen of blood gave three diagnoses:* one test indicated that too much thyroid hormone was present, the second test revealed that the concentration was just right, and the third test found that too little was present. When tests only create confusion, the patient's money should be saved.

A few words about the most recent test to appear. It is called the "radioassay for thyroxine" (a part of the thyroid hormone) and is supposed to be the latest improvement. The laboratory doing my blood work added this new test, without additional charge, to other tests being run. Otherwise, I would have missed a chance to evaluate this recent thyroid-function test. Forty-eight new patients with classical symptoms and physical findings of thyroid deficiency were tested. The normal range of results is considered to be 5.4 to 13.0 units. The lowest value among this group of patients was 6.0 and the upper limit was

14.1 units. Hence, there were supposed to be 47 normal individuals and one with too much secretion from the thyroid. The average reading for the group was 9.52 units. Interspersed with these new patients were 32 former patients who were being rechecked. All of them were on thyroid and were symptom-free. All of them gave values within the normal range by the new method. The average for the group was 9.09 units or identical with the group needing therapy. Had I followed the indication for thyroid function by the test and not started therapy on the new patients, I would not have made many friends and would only have influenced patients to change doctors. It is no surprise that patients from 49 of the 50 states have had to come to my office to get thyroid therapy denied them by physicians relying on thyroid-function tests.

A considerable amount of time has been spent in trying to find an objective indicator of thyroid activity. When using the Basal Metabolism test, it was always necessary to check the patient's temperature at the time of the test. If any fever were present, it would raise the metabolism about 10 percent for each degree elevation. Consistently the patients ran sub-normal temperatures, if no infection were present. For a century it has been known that the patient with too much thyroid activity runs a low grade fever, while the hypothyroid runs a subnormal temperature. The thought occurred that the patient could get a Basal Temperature with no cost or inconvenience. This work was published in 1942 and has been used continually since that time.[37] The thermometer is shaken down and placed by the bedside. As soon as the patient awakens in the morning after a good night's sleep, the thermometer is placed snugly in the armpit for 10 minutes. The normal range is 97.8 to 98.2 Fahrenheit.

This test is not specific for thyroid activity since starvation, an adrenal deficiency, or a pituitary deficiency will also give a low reading. These conditions are relatively rare and easily differentiated from a lack of thyroid. The temperature will usually rise a little with thyroid therapy, but it is not quantitative. If too much thyroid is administered, the Basal Temperature will go above normal in about 10 days. Hence, the test can be used as a guide in therapy. More will be said about this in the chapter on treatment. Needless to say, the profession has not been willing to adopt the Basal Temperature test. It is too simple, and no money can be made from it. Many physicians resent the patient trying to make a diagnosis on themselves. This practice is absurd,

especially in the thyroid-deficient patient for whom the diagnosis by the physician is so frequently wrong.

Laboratory tests have become big business. One of the leaders in the field markets six tests for thyroid function and informs the stockholders that it is a 45 million dollar business. Why do we need six worthless tests when a diagnosis can be made more accurately without any? Predictions are that in five years the thyroid function tests will amount to 100 million dollars for this company. The doctor is at the mercy of the salesmen who extoll the advantages of the different methods. This reckless waste of the patient's money is a big factor in the thrust for socialized medicine — a nightmare the public should be spared.

No disease is easier to diagnose than hypothyroidism, if one knows all the symptoms and the reliable physical findings. Combine these with the Basal Temperature, and few mistakes will be made. The physician and the patient as well, must be acquainted with the bizarre symptoms which may occur, or each may feel that this is a case of psychosomatic complaints.

CHAPTER VII

Tips on Treatment

No disease is easier to control than thyroid deficiency if the patient is intelligent and cooperative with the physician. Although the diagnosis can be made from the history and the Basal Temperature alone, it is well to have a comprehensive examination including a chest x-ray, a cardiogram, a urinalysis, a battery of blood tests and a physical about every two years for those under 50 years of age and yearly for those older. This type of examination picks up any other diseases in the beginning rather than after it is too late. Of course, anytime that some new complaint develops, this calls for a conference with the doctor at once.

A few things should be kept in mind in order to avoid trouble. The initial dosage should be small. A baby needing thyroid will tolerate one-quarter grain of desiccated thyroid daily until the age of 3 or 4 at which time the child may need as much as one-half grain. Between ages 6 and 12 one grain may be necessary, and above that age, the dosage can be adjusted as for an adult. Females in the teenage group — as well as adult females — can safely be started on one grain daily, while males of similar age and older, weighing more than 150 pounds, may require an initial dose of 2 grains. In the adult, if a previous heart attack has occurred, the initial dose should not exceed one-half grain, and a longer interval should elapse before the dosage is raised.

The initial dosage should be continued for at least a month after which time it is advisable to re-evaluate symptoms and Basal Temperature. If the symptoms have all subsided, the starting dosage may be continued indefinitely even though the Basal Temperature is still low. On the other hand, if some or all of the symptoms have persisted and the temperature is still low, the dosage may be raised by 50 percent for the next month. Re-evaluation is again made and the dosage adjusted as before. This procedure allows a gradual change in the physiology of the individual, and seldom will any difficulty be encoun-

tered. The patient with a previous heart attack should not be raised to more than 2 grains daily except in the hands of an expert with plenty of experience. Either male or female adults without heart trouble may take as much as 4 grains daily if needed. If symptoms still persist, some other cause should be sought for the symptoms.

What side effects can be expected? In the vast majority of cases there are none. *Thyroid is not a drug which is being taken, but a complement to one's normal hormone production that has been found inadequate.* If too much is administered, a new set of symptoms will appear such as nervousness, loss of weight in spite of a voracious appetite, profuse sweating, a rapid heart, inability to sleep many hours, yet too much energy when awake, and of most importance, an elevated Basal Temperature above 98.2 Fahrenheit — these call for cutting the dosage at once. Sometimes worry over finances, family problems, or a variety of other conditions may produce some of the symptoms of too much thyroid hormone, but the Basal Temperature will rule out everything else except some systemic infection. Rapid hearts are most often due to coffee, tea, chocolate, or caffeinated drinks.

There are a few individuals, probably not more than one percent of the total, who will feel worse on thyroid therapy from the start, yet they thought that they were half dead in the beginning. Zondek mentions these in his book, but in spite of his tremendous experience, he never understood why some patients failed to respond. Quite by accident I found that this same type of patient was also deficient in adrenal secretion. The use of 5 mg of prednisone daily, along with the thyroid, changes a failure into a resounding success. Cortisone has such a bad name from *over-dosage* in the treatment of arthritis that most patients shudder when they find that they need one of the adrenal hormones. Again, in this small dosage of 5 mg, it is not a drug but another of the hormones desperately needed. The combination of the limited dosage with simultaneous thyroid therapy has given no side effects over several years.

Which thyroid preparation should be used? There is a variety of preparations on the market, but the average physician depends upon information brought to him by the salesman who has a vested interest in his product. The company can make more money from the synthetic hormones; hence, the salesmen are pushing them with the pitch that they are "pure, more potent, better standardized, etc." I am amazed to see the number of patients who recently have been placed on thyroxine

when the physicians realized that thyroid therapy was needed. This product is as archaic as a one-horse-shay on Fifth Avenue. Obviously, the patient was not being relieved of the symptoms, or another doctor would not have been sought. The failure was due to the fact that thyroxine is only a part of the thyroid hormone; it is similar to wearing a blouse without a skirt. Thyroxine was isolated from the thyroid in 1917; it had some of the properties of the natural hormone. Many clinicians felt that this was the answer, and insisted that nothing else be used.

In 1949 another active compound, sodium liothyronine, was isolated from the thyroid gland; it was found to be 4 times more potent than thyroxine. Naturally the producer tried to convince the physicians that this was the product to use, and some doctors took the "bait." The new compound stimulates the heart more, and rapid beats are very common with it. Each of the synthetics will relieve some of the symptoms but not all of them. The next step was to combine the two synthetics into a single tablet; two such preparations are now available, one called Thyrolar and the other Euthroid. Each contains 4 times as much thyroxine by weight as liothyronine. Personal experience with both has indicated that when one of these synthetics is substituted in patients accustomed to the natural thyroid tablets, about 20 percent will experience rapid heart beats. There is probably a little too much of the liothyronine in the mixture; it may be significant that in Europe similar preparations have a ratio of 5 to one instead of 4 to one.

No one can guarantee that these two active synthetic compounds represent all of the physiological activity present in the natural gland. I have tried the various new preparations on patients who had been on the natural gland for years, and no one has preferred the synthetics. I repeatedly see patients who have been on one of the other preparations and still have some symptoms or physical findings which disappear on the whole-gland therapy. I consistently have used one of the standard brands of desiccated thyroid powder U.S.P. made into tablets. Since this is a research program, the same product has been used; it is obtained directly from the pharmaceutical laboratory, and is furnished to the patients. The present volume exceeds one million tablets annually.

Any of the standard brands of desiccated thyroid will be found satisfactory, but a word of caution about unmarked tablets. The manufacturer's emblem should be stamped on each tablet. Patients have brought in tablets without identification marks, purchased at the drug stores. Apparently the potency was subnormal, and symptoms were

not relieved. Substitution of a standard brand gave better results. It is suspected that the unmarked tablets were imported and had not been tested for activity.

The patient starting on thyroid treatment must look forward to a lifetime of therapy similar to the diabetic with insulin. If the medication is stopped, and yet no symptoms appear at once, the deposition of mucopolysaccharides will promptly begin, and the danger of a heart attack will increase. No one can predict when it will occur, but it seems definite that it will happen prematurely in the hypothyroid patient.

In the later years of life the requirement for thyroid hormone slowly decreases, and the dosage will have to be reduced a little. The Basal Temperature will help determine when too much is being taken. Senility and debility of old age have been markedly improved by thyroid therapy.

CHAPTER VIII

A New Look At Stress

Stress has been with man ever since Adam's rib was removed to make Eve. Surgery has long been known to cause stress. Stress is much like the weather: everyone talks about it, but efforts to control it are about as difficult as changing the weather. Stress is a broad term and hard to define. Here it will be considered as any condition arising from within the individual or in the environment causing a response in the defense mechanisms of greater magnitude than would be expected for normal existence. In the present discussion our primary concern is with the heart and the blood vessels.

Although the digestion of food causes the heart to beat faster and the blood vessels to carry more blood to the digestive tract, this exertion is necessary for existence and is not considered stress. Yet many heart attacks occur after a large meal indicating that even this physiological stress may be important. There are many ways in which stress can be shown to be detrimental, but in the present discussion we are especially interested in atherosclerosis. Stress is the fertilizer that insures a bumper crop, if the seed is planted in a susceptible soil. Stress does not start the process since people living to a ripe old age have been through an abundance of stress, yet may show very little evidence of atherosclerosis.

From whence comes the seed for stress and what makes the soil vulnerable? Curiously enough both seem to have a common origin. The earliest detectable chemical change in injured tissue is a rise in mucopolysaccharides. It matters little if the damage is due to mechanical trauma, to infections, to degenerative diseases or even to cancer. Mucopolysaccharides are markedly increased in atherosclerosis.

Next to the thyroid deficiency, stress is the most important factor in the genesis of atherosclerosis. It was the stress of war that caused the advanced degree of damage to the arteries of soldiers, first recorded in the Franco-German War in 1871, then in World War I, World War II,

the Korean conflict and even Vietnam. Stress was behind the four-fold increase in civilian atherosclerosis observed at Graz during World War II and discussed in Chapter one. The only premature deaths observed in the present study on patients treated with thyroid were under heavy stress at the time. The marked reduction in heart attacks with thyroid therapy would indicate that some stress is being reduced at the same time.

In the past the adrenal gland has held the center of the stage with stress. The time has come when we must add thyroid to the picture. This does not detract from the well established role of the adrenal, but it gives us a prophylactic approach to stress which the adrenal preparations can not do. The late Dr. Walter B. Caanon, one of our leading physiologists, in his work at Harvard showed that in an emergency the adrenal medulla (the interior part of the gland) gave a burst of adrenaline into the blood stream which immediately prepared the individual for fight or flight. The pupils of the eyes dilate, digestion ceases at once with the blood shifting to the periphery, the heart rate increases, the blood pressure goes up, the blood clots faster, the mental processes are stimulated and the individual has abnormal strength.

I can remember that, when I was only a small boy, the lightning struck my uncle's barn. His wife and daughters rushed out and pushed a wagon loaded with hay from the burning structure. The following day they could not budge the load. The adrenaline had given them abnormal strength during the excitement. At one time I was studying the cholesterol content of deer's blood. After catching the animal, the drawn blood would often clot before I could remove it from the syringe. The adrenaline of excitement had accelerated the clotting — one of Mother Nature's methods of reducing hemorrhage in case an accident causes a laceration.

Any type of excitement will call forth a burst of adrenaline from the adrenal medulla, but if the emergency lasts for more than a few minutes, the cortex of the adrenal (the exterior portion of the gland) begins to secrete more of its hormones. The latter are not so rapid in action, but their effect lasts much longer. Several hormones are produced in the cortex; they control mineral metabolism, aid in maintaining blood pressure, and aid in keeping a supply of sugar in the blood. The cortical hormones are manufactured from cholesterol; therefore it is not surprising that more cholesterol is stored in the adrenal cortex

than in any other tissue in the body. During stress, the cholesterol in the adrenal falls rapidly, but it is quickly restored after the stress is over.

Dr. Hans Selye, a scientist in Montreal, has spent many years studying stress.[38] He finds that there are 3 stages in the reaction of the adrenal gland: at the beginning of stress (a) an *Alarm Reaction* occurs at which time there are both an increased production and secretion of hormones. This stage is followed by (b) *Adaptation,* if the stress continues; during this period the demand is met by increased production of hormones. Finally, if overwhelming stress persists, the adrenal becomes (c) *Exhausted* and death follows. In the course of his experiments, Dr. Selye administered potent extracts of the adrenal cortex to experimental animals over prolonged periods. The animals developed hypertension, and some of the tissues showed changes resembling rheumatic fever or arthritis. It appears to Dr. Selye that degenerative diseases might be the result of chronic over-production of adrenal hormones, and he calls the alleged state "Over Adaptation." Occasionally, each of the endocrine glands does produce too much of its hormone for some unknown reason, but it seems unlikely that one gland would go berserk and produce degenerative diseases in the elderly.

A more plausible explanation is now possible for the results of Dr. Selye's prolonged administration of adrenal cortical extracts. In 1949 cortisone was isolated from the adrenal, and its synthesis gave unlimited supplies for experiments. It was soon learned that this compound would save many lives from stress and adrenal exhaustion. Injecting the compound for a few days gave the body a chance to recover. Arthritic patients "never had it so good"; pain and swelling disappeared, joints limbered up, and many patients could return to work. These results do not suggest that the arthritis had been caused by an over-production of adrenal-cortical hormone, but rather that there had been a deficiency of these valuable compounds. Unfortunately, the dramatic effects of cortisone soon wore off with the result that the patient was worse than before treatment. At first sight, this would fit into the "Over Adaptation" theory of Selye.

However, in 1950 a report from Hill at Harvard erased the confusion;[39] yet no one detected its significance and for 25 years it had been forgotten. He was not interested in arthritis, nor especially in the thyroid gland, but he investigated the effects of large doses of cortisone on thyroid function. He found that the production of thyroid hormone was markedly suppressed. In other words, he had created hypothy-

roidism by an excess of cortisone. Now we can understand why Selye had produced hypertension and arthritic changes with the chronic injections of cortical extracts. The thyroid deficiency led to the arthritis, just as Swain had demonstrated in 1929.[40] Menof reported in 1950 that thyroid therapy was efficacious in hypertension.[41] "Over-Adaptation" of the adrenal had not produced stress, but it had produced hypothyroidism which in turn had produced mucopolysaccharides described in chapter one. These compounds are present in all of our degenerative diseases resulting from stress.

There is no doubt that the adrenals are invaluable in combatting stress, but it seems obvious that stress can not be prevented by administering an excess of adrenal hormones. However, avoiding thyroid deficiency offers an excellent method for reducing stress.

Infectious diseases have been the bane of man's existence for centuries. Raising the resistance of the hypothyroid patient with thyroid therapy and avoiding the infection is far superior to allowing the infection to develop and using antibiotics to avoid death. *Repeated infections damage the lungs leading to emphysema, damage the heart leading to rheumatic fever, and damage the kidneys leading to uremia; yet thyroid therapy alone can avoid most of these stresses.* Repeated infections weaken the individual leading to arthritis in millions of patients. By treating children low in thyroid function, the deposition of mucopolysaccharides can be avoided, and the development of atherosclerosis and hypertension can be delayed. The atherosclerosis responsible for the complications of diabetes can be prevented by thyroid therapy. Thyroid deficiency is responsible for more stress than all other conditions known at the present time. For the prophylaxis of stress, begin with thyroid therapy when it is indicated.

So far, only environmental stresses have been considered, but of equal importance are those arising within the mind. Dr. Meyer Friedman,[42] a physician and investigator in San Francisco, has spent years studying this question. He has divided the population into two types; Type A is quite susceptible to heart attacks and is characterized by a personality with a drive to succeed, eagerness to compete, a desire for recognition, propensity for deadlines, and determination to accelerate physical and mental functions. Type B is the opposite in nature and is much less susceptible to heart attacks.

Dr. Friedman is correct in his division of the population, and many of both types have been encountered among my patients. The

fundamental question is, what makes the difference in the two groups and how can Type A be converted to Type B? The success in preventing heart attacks during the past 25 years merely by treating those susceptible with thyroid would indicate that Type A may be deficient in this hormone. Some of Friedman's data would confirm this suspicion. The cholesterols are higher in Type A and rise further during stress; a high cholesterol is common in hypothyroidism. The glucose tolerance test is somewhat reduced in Type A patients; this is a common finding in the patient lacking thyroid. Type A patients have a reduced tolerance for fats; this again may be related to thyroid function, as mentioned in Chapter 3 concerning Simonds work.[22]

Dr. Friedman decided that the Type A patient was not hypothyroid on insufficient data. He ran only a few patients for the serum thyroxine level and found no abnormalities. As pointed out in the chapter on thyroid function tests, the blood tests are unreliable, and furthermore, thyroxine does not represent all of the thyroid hormone. The therapeutic test of using thyroid therapy for symptoms of thyroid deficiency over many years and on thousands of patients with subnormal temperatures would seem to carry more weight than a fallacious blood test. The absence of premature heart attacks in this group of thyroid-treated patients leaves little to be desired. Without psychotherapy, the reduction in mucopolysaccharides seems to avoid the ravages of atherosclerosis. I might say that often the excessive drive of the Type A patients is lost without any deterioration in their ability. Their efficiency seems to be increased, and they can accomplish more without extra effort. Apparently mental stress, similar to that from the environment, is reduced by thyroid therapy.

One of the most controversial subjects on atherosclerosis is the role of exercise. The Europeans have walked more than the Americans, and have had a lower incidence of heart attacks. The physical culture enthusiasts credit exercise for the difference. A more plausible explanation would be the higher incidence of tuberculosis and other infectious diseases in Europe. This could explain the discrepancy. Work is stress and exercise is work. This should answer the problem, but it has not. It is well to remember that the laborers are not exempt from heart attacks. The pick and shovel workers do not enjoy greater health with long survival. They appear older than their years, and do not survive their peers. The athletes who must keep in excellent physical condition with maximum exertion are not immune to premature heart

attacks, as demonstrated by deaths on the football field and by two fatalities in professional wrestlers before the age of 50.

Each tissue in the body has a certain amount of reserve beyond that required for usual work and play. There is no evidence that we can increase that reserve with exercise. By graded training the heart can tolerate more punishment for longer periods, but has that added longevity to the heart or has it used up some of the reserve and shortened the life span? This is a difficult question to answer, and no controls can be run. It is true that following a heart attack, if death does not occur, some new vessels may open up, and the individual may tolerate more exercise than he did before the attack. This does not mean that more reserve has been added to the heart muscle. The blood supply has been shifted and another area of the muscle is now in jeopardy if another emergency occurs. It is not recommended that the patient should spend the rest of his life in bed just to prolong life. That would not be living. But it does signify that he should not try to become superman, or he will soon use up his reserve.

A few years ago jogging became the rage. From the beginning I took a dim view of this attempt to strengthen the heart. It would not harm healthy individuals, but why should it help them? On the other hand, those who needed it the most had a reasonable chance of being damaged with it. Some of the older members of society did not have that much reserve left in their hearts, and should develop a heart attack as a result of the additional stress. It happened. More lives were lost than were benefitted and jogging is no longer advised by those seeing the results.

Now a new procedure for improving health is being advocated by some very competent physicians. It is advertised over the radio and TV with great promises for benefit. "Check your heart on the treadmill or exercise machine and determine your tolerance." The danger of such a procedure is admitted by the fact that it must be done in the hospital with one or more doctors in attendance and requires 2 hours. In the *Journal of the American Medical Association* for February 23, 1976 a fatal case is reported. The 56-year-old male was admitted for removal of the prostate. Although the electrocardiogram was normal he was given the exercise test which again showed no evidence of heart trouble. The exercise was stopped when his heart rate reached 150 per minute. Throughout the test the cardiogram was normal. Following the test the tracings were repeated at 2-minute-intervals, and all were

normal. The patient was returned to his room. About 30 minutes later chest pain developed, he collapsed and died with a typical picture of a heart attack. The autopsy revealed a very recent total occlusion of one of the coronary arteries. This is not the first such case to be reported, and it is apparent that the electrocardiogram is not infallible. There is no difference between the treadmill and jogging except the ambulance does not have to pick up the victim. The cost of the treadmill test ($80-$150) seems excessive for the opportunity of committing suicide.

Nature gives us warning when we have over-stressed. Curtailing activity a little at the time is more reliable than laboratory tests. As one grows older it is prudent to gradually reduce one's work as well as exercise. If playing a strenuous game of tennis appeals to you, that is your privilege; however you should give it up when you are made weary by it unless you want to do some heart damage. *One should exercise for pleasure, but dismiss the idea that it will prolong life.* My most remarkable patient exercised only by lifting his pencil. He was an accountant still working at age 80, was alert and spry. No doubt exercise would have killed him at a much younger age. Stress is the greatest accelerator of atherosclerosis, have respect for it.

Another mystery about heart attacks has been the higher incidence among men. For a time it was felt by some investigators that the female sex hormone was protecting the "weaker" sex. Attempts were made to treat men recovering from the first heart attack with the female hormone. Two things happened: (1) the men developed breasts and lost their potency, (2) the second heart attack occurred earlier than expected. The deleterious effect of the female sex hormone on males was clearly demonstrated from another angle. The Veterans Administration used estrogen in treating a large series of men with cancer of the prostate, and found that the growth of the cancer was retarded. However, the men died from heart attacks sooner than they would have from cancer. Excess of female hormone creates a stress in the male.

For many years it has been continually demonstrated that females have more resistance to infectious diseases than males. This does not give support to the thesis that health can be built up with exercise. If only tuberculosis is considered, the excess of deaths among men is almost identical with the excess of heart attacks among males. It was pointed out in Chapter one that the rise in heart attacks today has been a result of reducing the deaths from tuberculosis. If this is true, the

excess of males formerly afflicted with tuberculosis could account for the excess of heart attacks among the stronger sex. But why should the males be more susceptible to infectious diseases? Certainly the stress of exercise must be considered among the possibilities. Until the age of puberty there is no difference between the sexes in the incidence of fibrosis of the heart muscle. Fibrosis indicates damage to the muscle fibers as a result of various diseases. While the girls are assisting their mothers, the boys are engaging in competitive sports, fighting, and building up the muscle-mass to become He-Men. From that time on there is more fibrosis in the heart muscle of the male. This extra stress could be a factor large enough to account for the excess of both tuberculosis and heart attacks in the alleged "Stronger Sex." What Price Glory! *Women's Lib may be their greatest achievement, but if the extra stress to meet the competition lowers their vitality, it may be their darkest hour.*

CHAPTER IX

Prevention: The Key to Progress in Medicine

It would appear that heart attacks may not be cured, but they can be prevented — at least those occurring early in life can be delayed until advancing years when other degenerative diseases have an opportunity to enter the competition. The rapid rise of heart attacks in the 20th century was not due to changes in the environment, such as cigarette smoking, too much animal fat in the diet, a lack of exercise, etc. It was the result of the conquest of infectious diseases by improved medical care and antibiotics which prolonged the lives of over half of the population. It is obvious from previous pages that those individuals susceptible to infections are likewise susceptible to atherosclerosis; prolonging their lives permits them to live long enough to develop heart attacks. At the present time, natural consequences have a far greater influence on public health than have environmental factors.

In reviewing the history of the scourges of mankind, one finds that the prevention of smallpox changed the leading cause of death from this killer of young children, to tuberculosis since the population was permitted to live longer. Then when tuberculosis was deterred, a new champion of death had to emerge. At the turn of the century, if a meeting of the minds had been held to predict which disease would come to the fore in case infections were conquered, no one would have mentioned "heart attacks" — they were not as yet in our vocabulary. When heart attacks did appear, they were so frequent that they shook the halls of science at their very foundations. Had another plague arrived to decimate mankind? The researchers failed to realize that the average age of survival was climbing rapidly. The health of the nation was the best in history.

The solution to THE RIDDLE OF HEART ATTACKS again came from preventive medicine. Because of a thorough knowledge of the physiology learned from teaching the subject for five years and

demonstrating to university students the effects of the removal of the thyroid glands in baby rabbits, some of the problems in medicine were solved. Patients highly susceptible to infections were similar to the thyroidectomized baby rabbits. Thyroid therapy raised the resistance, and many infections could be prevented. Women with menstrual or reproductive problems had some of the same symptoms as the baby rabbits. Thyroid therapy relieved many of the problems in the human. Fatigue and lethargy were characteristic of the baby rabbits. Patients with these symptoms responded dramatically to thyroid administration. When it became obvious that elevated cholesterols were often present in adult patients with heart attacks, it was recalled that the baby rabbits had high cholesterols. Several investigators had found that thyroid therapy would lower the cholesterol level in the blood.

The prevention of infections, of menstrual disorders, of fatigue, and many other ailments with thyroid therapy continued in my patients for 13 years. Suddenly it was realized that none of the treated group had developed heart attacks. It was only then that the idea arose that thyroid deficiency might have a role in atherosclerosis and heart attacks. Searching the literature revealed some astounding facts. In the last century, long before heart attacks were understood, marked atherosclerosis was reported in an autopsy on a patient having no thyroid gland. Furthermore, removal of goiters to prevent suffocation was followed by accelerated atherosclerosis. Also, removal of the thyroid from animals led to damage of the arteries carrying blood to the heart. Administration of thyroid hormone to these animals prevented the arterial damage. Thyroid therapy had been found life-saving during World War I in patients with heart disease and low thyroid function. All of these observations were known before heart attacks had become numerous, but they had been forgotten.

It was in 1950 that a program was started in a busy general practice to see if detecting all of the thyroid deficiencies and treating them with thyroid would prevent premature heart attacks. After 25 years of this regime, there can be no doubt that thyroid therapy alone now offers more hope for reducing the devastation of heart attacks than all other methods combined. This was accomplished only from the practice of preventive medicine and without any financial aid from the government or private research sources. The results indicate that any physician without special equipment or without hospitalization of his patients for special studies can reduce the number of premature heart

attacks by 94 percent. We owe a huge debt to the baby rabbits that gave their lives that man might live.

Unfortunately, some other rabbits started a different train of thought and implicated cholesterol in atherosclerosis. This has delayed progress for 60 years, but the mistake is explained in the final chapter of this short treatise.

CHAPTER X

The Demise of the Cholesterol Theory

In science, theories are created to explain the correlation of some known facts and to assist in designing experiments for the collection of more pertinent data. Very often the collection of more facts makes the theory untenable, and it must be abandoned. The cholesterol theory was born to explain the rapid rise in heart attacks in the 20th century. Many facts, present before the theory arose, were not explained by it, and many new facts, accumulated during the past 25 years, fail to support the theory. *The time has come to bury the theory that cholesterol causes heart attacks; some of the deeds of this shady character preclude a funeral with "full military honors" or burial in one of our National Shrines.*

A few words about the facts upon which the theory was founded are in order. The first was the presence of a high incidence of cholesterol in atherosclerotic blood vessels. This is always true, but as early as 1858 Virchow,[43] the father of modern Pathology, clearly showed that cholesterol did not start the process but that it was the end product of degeneration. Damage to the tissue became evident first, then came an accumulation of fat, and finally, as the scar tissue was formed, a high content of cholesterol appeared. It was part of the healing process of a wound from damage caused by something else. This sequence of events was confirmed by Leary,[44] a modern Pathologist, at Harvard. Hence, the first evidence that cholesterol causes heart attacks must be discarded.

The second major evidence incriminating cholesterol was the scarcity of heart attacks in the underprivileged countries whose populations eat relatively little animal products containing cholesterol. This fact is well established, but does it prove that the absence of cholesterol was responsible? Absolutely not, for, as noted in Chapter one, Strong[4] found atherosclerosis in the arteries of all of the children by the age of three coming to autopsy in the underprivileged countries as well as

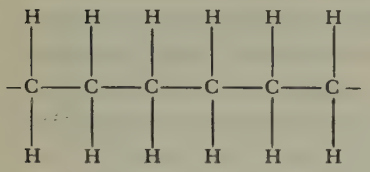
in meat-eating countries. In fact, Higginson[45] and others have found more atherosclerosis in the younger Bantu than is found in America. Then why do the Bantu have such a low rate of heart attacks? Again, in Chapter one the answer was pointed out from the autopsies at Graz, Austria. In countries with a high incidence of deaths from infectious diseases, patients die from infections at an early age before heart attacks can occur. Proof for this among the Bantu comes from a report by Laurie[46] who found a rapid rise in heart attacks as tuberculosis declined. The second evidence against cholesterol now has a more plausible explanation.

The third and final proof that cholesterol was responsible for heart attacks came from Europe during World War II. Heart attacks that had been rising rapidly fell precipitously during the war when cholesterol-containing foods were unavailable. As mentioned in Chapter one, autopsies proved that heart attacks fell as much as 75 percent, but atherosclerosis increased four times in severity. Tuberculosis exploded; patients were dying from it before heart attacks could occur. The absence of cholesterol from the diet had offered no protection to the patients' arteries. The most potent evidence for the theory must be discarded in view of the facts.

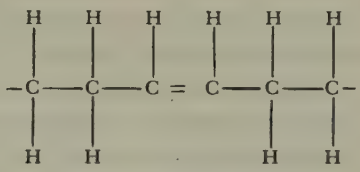
It is apparent that the cholesterol theory has become a myth causing a tremendous loss in time and money. But that is not the most serious part of the story. Evidence has been accumulating that grave damage has been done by a shift in diet trying to prove the cholesterol theory. From time to time, hard water, sugar, coffee, and a host of other ingredients in our diet have been accused of causing heart attacks, but they have received little attention in comparison to that devoted to saturated fats. Saturated fats are more abundant in the animal world and unsaturated fats predominate in the vegetable world and in fish. This has been part of nature's adaptation to the environment, and does not signify that a mistake was made in the creation of warm-blooded animals. There is no cholesterol in the plant world, yet no animal can survive without it. In man, the brain and spinal cord make up only 2 percent of the body weight; still 23 percent of the cholesterol is in the central nervous system. If for eons man were to feed on food low in cholesterol, he might wind up with the brain of a jellyfish.

The layman may understand the dangers of eating too much poly-unsaturated fat if a few words are devoted to the difference between the saturated and the unsaturated varieties. They differ only in the

amount of hydrogen in the molecule. Both are composed of long carbon chains as illustrated here:



Saturated Fat



Unsaturated Fat

In unsaturated fats there is a double bond uniting two carbon atoms. Since carbon has only 4 bonds to unite with other elements, there is nothing to hold the hydrogen to the carbon at the double bond. This double-bond linkage is relatively unstable and tends to break apart into two smaller molecules either in the laboratory or within the body. The double bond is similar to cutting a notch in a stick. Any attempt to bend the stick may cause a break at the notch. If the fat breaks up into two sections the carbons on either frayed end have a powerful attraction for any other compound with an opposite charge. *Some of the compounds formed from the breakdown of unsaturated fats are toxic to the body. Some of them have been demonstrated to form cancer when injected into or fed to animals.*

Normally the saturated fat molecules are oxidized or burned only at the end of the chain, two atoms of carbon being eliminated each time. There are no toxic compounds created under these circumstances. However, the unsaturated fats have a weakness for oxidation at the double bond. Two unnatural carbon linkages are available if the chain breaks at this point. If there are two or more double bonds in the particular fat (polyunsaturated), the increased number of notches on the stick weaken it further; accordingly, there are more chances of liberation of abnormal carbon atoms with the subsequent formation of toxic compounds. Is this theoretical and academic? Far from it; evidence, though fragmentary, clearly indicates that polyunsaturated fats cause more toxicity than those with only one double bond.

Were diets high in unsaturated fats tried on animals before the demands were made that the human diet be changed? Absolutely not. The theorists were so sure the world was coming to an end from heart attacks that there was no time to wait for experimental proof of their

theory. If this sounds like a fairy tale, note the following which occurred as late as 1970. At a symposium on "Diet and Cardiovascular Disease" refereed by Frederick J. Stare, M.D.,[47] an outstanding nutritionist from Harvard, it was concluded that — the computed cost in terms of human lives alone of waiting for ultimate proof for the prevention of coronary disease by lowering the serum cholesterol with diet is up to one million lives in the next five to seven years in the U.S.A. alone —. In the same article, dangers of such a diet, although acknowledged, are dismissed without consideration.

It is obvious that the theorists have totally disregarded the fact that the average age of survival has continued to climb all during the rise in heart attacks and is now the best in history. Their obsession for diet is often motivated by vested interests of the manufacturers of unsaturated fats with their exaggerated claims without foundation. The latest colossal mistake is the circumcision of the yolk from the egg. Egg yolk contains one of the unsaturated fats essential for growth and health. Its necessity has been proven without reasonable doubt; no one has demonstrated any harmful effects from the cholesterol in egg yolk which is being eliminated from the diet.

If the literature is scanned for evidence of toxicity of unsaturated fats in the diet, one report precedes the cholesterol theory. In 1945 Rausch[48] at the University of Wisconsin noted that adding corn oil to the diet of rats increased their susceptibility to tumors. This observation alone should have called for a thorough investigation of this group of compounds before they were applied to the human. Perhaps the cholesterol activists considered unsaturated fats safe since cancer among the underprivileged populations had been very low. It is well to bear in mind that the reason is identical with the scarcity of heart attacks — most of the people die from infections before they reach the age at which cancer or heart attacks might be expected.

It is a sad commentary on the American scientist that the potential danger of unsaturated fats had to be brought to their attention by an abnormal number of cases of cancer in patients receiving diets high in unsaturated fats for the prevention of heart disease. In 1971 a report by Pearce[49] appeared in *Lancet,* vol. 1, page 464, entitled, "Incidence of Cancer in Men on a Diet High in Polyunsaturated Fat." Why was this article sent to England where unsaturated fats have never been popular? Was it refused publication in American journals? It seems strange, but regardless, there has been an attempt to sweep this infor-

mation under the rug. First, however, what were the details? Soon after the diet alterations were suggested, a controlled study was undertaken at the Veterans Hospital in Los Angeles. A total of 846 men were matched and divided into two groups; one received the customary diet including animal fats, the other group was fed the same quantity of polyunsaturated fats. Over a period of 8 years, careful records were kept including autopsies on the majority of deaths. There were a few more deaths from heart attacks among the men on the saturated fats, but variations from this disease are not unusual. Of far greater importance, there were almost twice as many deaths from cancer in the group on polyunsaturated fats. This came as a surprise, for deaths from cancer do not show the variation in incidence that is seen with heart attacks.

In the previous volume of *Lancet* there appeared a letter to the Editor from Harman,[50] an investigator from the Veterans Hospital in San Francisco. He cautioned against the use of polyunsaturated fats in human diets because of the cancer-producing compounds that can result from the breaking of the double bond, mentioned above. He also pointed out that the Japanese have a much higher incidence of cancer of the stomach than is seen in other countries. He suggested that the high content of polyunsaturated fats in the fish diet of the Japanese might be a major factor in the incidence of cancer. He further stated that some reduction in the breakdown of unsaturated fats could be obtained with an increase in Vitamin E in the diet, since it is an anti-oxidizing agent.

The last statement is probably well-founded. Vitamin E was seldom used before the introduction of unsaturated fats in abnormal amounts. At the present time, it is suggested for everything from falling hair to fallen arches, and if one is eating an excess of polyunsaturated fats, any tissue in the body might be poisoned without it. It seems foolish to administer a poison requiring an antidote for neutralization; it would appear more logical to avoid the unsaturated fats in the first place.

A few publications on experimental animals began to appear after these reports. In 1960 Ershoff[51] found that fish oil used as 10 percent of the diet in growing rats stopped growth and produced diarrhea. Vitamin E was found to be beneficial. In 1967 Norkin[52] reported more cirrhosis of the liver in rats fed corn oil than in those fed a saturated fat, coconut oil. This undesirable effect could be prevented with extra

Vitamin E. In 1968 Carrol[53] in Canada produced more mammary cancers in rats fed corn oil than in those fed coconut oil. In 1968 in the *New England Journal of Medicine* Ritchie[54] reported seven premature babies suffering from edema, hemolytic anemia, and abnormal blood cells. Because of the propaganda about saturated fats, they had been started on pre-diluted, skimmed cows' milk, vegetable oils and iron. They responded to large doses of Vitamin E. Some common sense, with the old-fashioned formula using milk-fat, no doubt would have prevented this near tragedy.

Now 19 years after the warning that unsaturated fats might cause cancer, polyunsaturated fats are being thrust upon the public by the press, radio and TV. Similar evidence from a pesticide, a food additive, or a pollutant in the environment would call for immediate action with cessation of the use of the suspected item until it was proven safe. Nothing has been done about the potential danger of unsaturated fats except some feeble attempts to discredit the observations from Los Angeles. There have been four other reports of somewhat similar nature although fewer patients were used and the interval of study was shorter. These were reviewed by Ederer[55] in 1971. One report from Oslo confirmed an excess of deaths from cancer in the patients on unsaturated fat diets. The three other studies — one from Finland, one from London, and one from the University of Minnesota — showed no difference in the incidence of cancer in the experimental and control diets. It sounds like the World Series with the best three out of five trials showing no effect. Ederer concluded from a statistical treatment of all five studies that it is unlikely that cancer is a hazard in the dietary treatment of heart disease. He does admit that the patients in the Los Angeles study were 20-25 years older than those in the other four studies. This may well be the important part of the argument. Cancer is a disease of older age groups, and studies on young patients in no way can be compared with those on older populations.

Another report recently incriminates unsaturated fats in cancer. Dr. Mackie[56] of Sydney, Australia, reported five cases of malignant melanoma (pigmented cancer) appearing in a matter of 12 weeks. This highly fatal disease is quite rare; five cases constitutes an epidemic. All of the cases were in sunbathers exposed to ultraviolet light. Cancer of the skin is common in farmers and fishermen, but melanomas seldom occur. Each of the five cases had shifted their diets to polyun-

saturated fats. Dr. Mackie felt that this was a factor in causing the rare disease.

It will take a tremendous number of cases and considerable time to test the role of unsaturated fats in the origin of cancer. There is no doubt that an increased incidence has occurred in experimental animals. Why have not more studies in animals been undertaken? Finally the American journals are beginning to publish articles on the dangers of unsaturated fats. An editorial by Pinckney[57] in the June 1973 issue of the *American Heart Journal* reviews evidence that the unsaturated fats hasten aging of the skin, are toxic to both animals and man, and furthermore, that the use of such diets has not prevented heart attacks.

It is apparent that the use of polyunsaturated fats in concentrations of 10 to 15 percent as recommended by some of our scientists is highly controversial. There is no concrete evidence that this alteration in our diet over a period of 25 years has benefitted our health. There is positive proof that the polyunsaturated fats are toxic to experimental animals, and additional Vitamin E must be added as an antidote if reasonable quantities of such fats are eaten. A single report on premature babies indicates that the human may also be poisoned by this concentration of polyunsaturated fats. Both babies and oldsters are known to be more susceptible to medicines and toxins than are young adults. The suggestion from two independent reports that the aged, fed polyunsaturated fats, may develop more cancer cannot be dismissed. If this type of diet has no demonstrable benefit, WHY SHOULD IT BE USED?

The American Cancer Society was shocked at a 5 percent rise in cancer the first seven months of 1975 when a one percent had been anticipated. In the frantic search for causes of the rise in cancer certainly the increase in polyunsaturated fats for the past 25 years cannot be neglected. The housewife followed the scientists' advice 25 years ago when the circumstantial evidence indicated that heart attacks might be avoided by crowding in more polyunsaturated fats and avoiding animal fats. Now it is apparent that the saturated fats were not causing the rise in heart attacks. This change in the death pattern was caused by eliminating infectious deaths allowing a NEW POPULATION to reach an age capable of developing heart attacks. *The evidence that the polyunsaturated fats are toxic and may cause cancer is not theoretical; it is far more conclusive than the evidence that saturated fats were causing heart attacks.*

It is time for the housewife to make another decision. Can she afford to continue the unsaturated fats with their demonstrated toxicity and run the risk of cancer in her family? It was difficult to deny the members of the family their eggs, bacon, etc., but mothers have a way of achieving a desirable change. Now she has found that a mistake was made and she should be just as eager to reverse her stand and prevent some new tragedies. It will be hard to ignore the propaganda that the saturated fats cause heart disease. That propaganda will stop abruptly when the housewife passes up the unsaturated fats and fills her basket with cream, butter, eggs, lard, fat meat, and the other goodies which the family has been craving. The propaganda for unsaturates is perpetuated only by the vested interests of the manufacturers. The few pennies saved on the budget by purchasing cheaper margarines and oils may be a poor investment compared to the prolonged and horrible death from cancer. If the polyunsaturated fats are safe, let the manufacturers prove it on animals before a new plague develops from the false statements that unsaturated fats will prevent heart attacks.

Everyone should have the privilege of playing Russian Roulette if it is desired, but it is only fair to have the warning that with the use of polyunsaturated fats the gun probably contains live ammunition.

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